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Listerian Oration.¹

THE SEEDS OF TIME: THE IMPACT OF MICROBIOLOGY ON HUMAN AFFAIRS SINCE LISTER'S DAY.

By SIR MACFARLANE BURNET,
Melbourne.

I AM deeply honoured by your invitation to deliver the Listerian Oration for 1952, but as on all such occasions I have found difficulty in deciding what is an appropriate subject for the purpose and how it should be treated. Should one on such an occasion as this present an account of one's own current work necessarily of interest to only a small minority of specialists or should one attempt a broader picture and run the risk of being pretentious, platitudinous and dull? I think perhaps the choice should be made not so much by a consideration of what the audience would appreciate but by what the lecturer feels is the subject on which it is most desirable that he should clarify his own mind. The only way to learn a subject is to write a book about it; a lecture is the next best. It seemed to me, then, that I knew less than I should about the history of my own subject and that there was a real story to be told of the impact of microbiology on human life over the past hundred years. Lister's professional life began just a century ago at a time when microbiology had no visible bearing on human affairs but at least when looked at in retrospect was clearly pregnant with important consequences.

¹ Delivered at a meeting of the South Australian Branch of the British Medical Association on April 24, 1952.

I have taken the title of this lecture from Macbeth: If you can look into the seeds of time And say which grain will grow and which will not... These two lines seem to me to express almost perfectly the justification for research, and if we remember that they were spoken by Banquo to the three witches we can read into the words a sense of the menace as well as the promise of every major advance in human knowledge. My theme tonight is that in the last one hundred years applied microbiology has been the most important factor in the social and political developments of our time; that it has made it possible for virtually every child born healthy to look forward to the normal span of life and in the process changed the whole biological structure of our species. Starting with a picture of the state of microbiology when Lister was a young man, I should like to sketch in the broadest outline the way it has developed to the present day, and to end with an attempt, however feeble, to "look into the seeds of time" today.

The Scientific Scene in 1852.

In 1852 Joseph Lister was a young man who was just about to move from London to Edinburgh to become Syme's house surgeon. It is not known that he had yet begun to think for himself about the problems of surgical infection, but we can be quite sure that at University College Hospital he had already had plenty of experience of hospital sepsis. Already one man, Semmelweis, had clearly realized where the danger lay in surgical and lying-in hospitals. In 1847 he had correctly deduced the essential nature of puerperal infection and had reduced the mortality rate of his obstetric patients from 25% to 1% by antiseptic procedures based on the use of chloride of lime solutions. No one in Europe paid any attention to his work until the same conceptions had been developed by Lister twenty years later. As in so much of the history of science and medicine,

the man too far ahead of his time is ineffectual. It is hardly too much to say that no major discovery destined to be fully incorporated into established knowledge and techniques is made more than a year or two before it is inevitable. The stage for the acceptance of Semmelweis's discovery had not been set in 1852. This is evident when we look at the scientific and medical scene in that year.

In Victorian England the Great Exhibition had just been held and there was a feeling in the air that applied science was initiating an enormous increase in industrial prosperity. A social conscience was arising. Farr and Chadwick had begun objective statistical studies of how the masses of the people lived and when and how they died. The Crimean War was looming—Florence Nightingale was obtaining her first practical experience of the problems of nursing in 1852. Soon she would be in a position, as a result of her work at Scutari, to act as a gadfly to administrators and governments for the improvement of hospital care, first of the soldier, then of the common people.

The stream of research in microbiology was still only a trickle, but the interest and the points of view were developing that foreshadowed the sudden emergence of bacteriology with Pasteur and Koch. Pasteur had completed his chemical work on the relation of crystal form to the rotation of polarized light, but had not yet moved into the microbiological field. The controversy in regard to spontaneous generation of microorganisms was gathering force and ingenious experiments of the type made familiar by Pasteur's work a few years later were already being performed. The method of sterilizing air by filtering it through cotton-wool was to be discovered in 1853. Many people were interested in the zymotic theory of infectious disease. The fungal cause of ringworm was recognized by Schölein in 1839 and Bassi's work on the muscardine disease of silkworms about the same time had shown that microscopically visible fungi could produce a fatal disease. Microscopists were already looking at blood and discharges from diseased patients and animals in the hope of seeing microorganisms. It is probable that in the year we are considering two French microscopists actually saw the anthrax bacillus in the blood of sheep dying of the disease. Two sets of phenomena, alcoholic fermentation and the putrefaction of dead animal matter, whose further study was to have great significance, were centres of major controversy with Liebig, who held firmly to the view that they were wholly chemical processes against the growing evidence that living microorganisms were the effective agents.

It is fair to say, then, that looking back on the situation in 1852 one can see the first differentiation of the lines along which microbiology was to develop. The experimental method was now being applied to biological problems by men familiar with the rapid progress that chemistry had made in the first half of the nineteenth century. There was still work to be done before the time was ripe for the sudden clarification of the nature of infectious disease that came with Pasteur and Koch, but the movement of microbiology had already developed an impetus that made its continuing development inevitable.

The Principles of Applied Microbiology.

I cannot of course make any attempt to do more than outline the subsequent progress of microbiology. It may be useful to look at its development from the point of view of the emergence of the principles on which present control of microbial infection is based. There are five important principles to be considered and I shall say a few words about the way each of these emerged.

(1) *Infectious Disease is Due to the Multiplication Within the Body of Specific Types of Microorganism.*

The doctrine of the specificity of infectious disease was established primarily from studies of anthrax and of the septic infections induced by the injection of putrid blood and similar materials into laboratory animals. A long series of investigations by others led up to Koch's two outstanding contributions in these fields in 1876 and 1878. Once these two papers had been published, bacteriology became a science and with the development also by Koch

of staining techniques and methods of plating bacteria on solid media by 1881 it took on almost its present form. It must not be forgotten, however, that long before Koch's work clinicians and epidemiologists, Bretonneau, Henle, Budd and Snow, had correctly inferred that diseases like diphtheria, typhoid fever and cholera were specific entities each with its characteristic self-reproducing aetiological agent.

Once Koch's methods had been established the task of isolating and characterizing the causes of bacterial disease became relatively straightforward. Very often, of course, the picture soon appeared much more complex than was the case with anthrax or tuberculosis. Staphylococci, pneumococci, streptococci and the Salmonellas all produce human disease and in a very broad way it can be said that each form gives rise to a fairly characteristic pathological condition. Yet each comprises many different forms with a very wide range of immunological character. Only in a very restricted sense can one regard the principle of specificity as being exemplified in such infections. It is interesting to look at the frequency with which it has been necessary for the advance of science that provisional generalizations should be regarded for a period as representing established truth—although subsequent work shows them to be false. It would have confused the progress of bacteriology if the complexities of serological races of pneumococci had been discovered too early, just as a knowledge of isotopes would have introduced a completely indigestible element into nineteenth century chemistry.

(2) *Each Type of Pathogen has its Own "Natural History" and by the Understanding of How the Organism Survives in Nature and Manages to Reach New Susceptible Hosts it is Often Possible to Break the Cycle.*

The ecological approach to infectious disease came rather slowly and more as an application of common sense to each problem as it arose than from any attempt to generalize. The work of Budd on the spread of typhoid fever and of Snow on cholera indicated the part played by water in carrying infection of certain types long before the responsible microorganisms had been isolated. Many of you will have read the story of the Broad Street pump in Soho from which 500 deaths from cholera resulted in August, 1854. Snow's incrimination of a single source of water-borne infection provided the prototype of a hundred later epidemiological investigations, and pointed clearly to simple methods of preventing one important group of diseases.

Perhaps it will give some picture of the hygienic conditions of the under-privileged just one hundred years ago to tell briefly the story of the Broad Street pump. It is perhaps the most famous of all epidemiological investigations, and in the history of bacteriology Snow's report on this epidemic of water-borne cholera played an important part both in establishing the doctrine of specificity of infectious disease and in indicating that fecal pollution of drinking water was the major cause of intestinal disease.

Cholera had been prevalent to some extent in London since 1848, and cases had been occurring in the Soho district in small numbers during August, 1854. Then on the afternoon of August 30 an explosive and intensely lethal outbreak of cholera began. Next day there were 31 deaths and on the days following 131, 125, 58, 52 and 26. There was of course panic, a disorganized exodus from the district and overcrowding of the hospitals. Florence Nightingale served as a volunteer nurse at the Middlesex Hospital where it is stated that "the authorities were obliged to send out their usual patients in order to take in cholera patients from the Soho neighbourhood. . . . Many of them were fallen women of the district—the disease taking worse hold of them than of any. Miss Nightingale was up day and night undressing them, putting on turpentine stups etc. herself, to as many as she could manage."

The investigations of John Snow clearly incriminated the water from what was called the Broad Street pump, and on September 8 the handle of the pump was removed by the local authorities. Actually it appears that the water of

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pump ceased to be infectious on or shortly before September 3.

In the 1850's London water was supplied by a large number of water companies, often so highly competitive that two companies would have pipes running down the same street. Practically all drew their water from the Thames and though filtration and storage were attempted by some of the companies they were grossly inefficient. A large part of Snow's studies was applied to showing that the Southwark and Vauxhall Company's water produced 71 fatal cases of cholera per 10,000 houses supplied while the Lambeth Company's water was responsible for only five.

Company's water was not particularly attractive and many people preferred water from local wells. There was a widow in Hampstead, for instance, who so greatly preferred the water from the Broad Street pump that she had a carter bring a bottle to her daily. It had the reputation of being clear, sparkling and cool, but the widow died of cholera on September 2. The pump drew water from a shallow well 28 to 30 feet deep into which percolated surface drainage as well as the fluids which leaked out from some of the innumerable cesspools that existed in the London area. This particular well was a few yards from a main sewer and the disintegrating brick wall of a cesspool was only three feet six inches from the well. Snow suggested that the main source of infection was from an infant who was ill in the house served by the cesspool in question on August 28.

The distribution of those who were infected and those who escaped in the first explosive phase of the epidemic was almost strictly correlated with whether they had or had not used the well water. The picture is very similar to that of any water or milk-borne epidemic. There were the usual dramatic contrasts. A factory in Broad Street had 22 deaths but a brewery also in Broad Street escaped, presumably because the workers had no interest in drinking water.

I might add that in 1946 I tried to find Broad Street and the site of the pump, but the streets of Soho have been greatly changed since 1854 and Broad Street no longer exists and there is not even a plaque to mark the site of the infamous pump.

The greatest stimulus to ecological studies of disease came with the recognition of the carriage of infection by insects or other arthropods. Like so many other discoveries, this was preceded by speculations in regard to malaria and yellow fever, but first came into the general stream of microbiology with Theobald Smith's recognition of the part played by ticks in the cattle disease Texas fever. In 1898 Ross showed that bird malaria was transmitted by a Culicine mosquito and within the next two years Grassi made the necessary extension to human malaria by demonstrating the role of *Anopheles* mosquitoes. These discoveries were almost immediately followed by the demonstration that yellow fever was also a mosquito-borne disease. A concentration of work on these two major scourges of the tropics soon showed the enormous importance for disease control of a full understanding of all details of the habits and preferences of the vector species. The development of malaria control methods probably did more than anything else to establish the ecological approach to entomological and other general biological problems. One general feature of infectious disease control is well exemplified in malaria. This is the desirability of attacking at more than one point in the cycle. If all malaria-carrying mosquitoes can be eliminated from a country there will be no malaria, but in general no absolute control can be reached except by long-continued effort. In the intervening period such diverse measures as rendering breeding places unsuitable for larvæ of the significant anophelines, residual DDT treatment of the indoor resting sites of mosquitoes and chemoprophylaxis by "Atebrin" or "Paludrine" plus many others may all be adopted.

(3) Microorganisms Can be Killed by Various Physical and Chemical Methods, Sometimes by Methods which Do Not Damage the Host's Cells.

The destruction of bacterial activity by heat or chemical agents and the application of such methods to the prevention of infection may well take its origin from the kitchen.

Food can be preserved from putrefaction by the addition of sugar, salt or vinegar in concentrations high enough to be bacteriostatic, and at the beginning of the nineteenth century the French confectioner Appert described his method of preserving food by heating it in clean sealed bottles to the temperature of boiling water. As a by-product of the controversy on spontaneous generation many ingenious methods were devised of heating apparatus, nutrient fluids and air to destroy preexistent micro-organisms before the crucial experiments were set up. Out of this work there gradually emerged a recognition of the conditions needed to destroy bacterial spores and a technique that could readily be applied to the requirements of bacteriology and of aseptic technique in surgery.

Lister came to his work on surgical antisepsis at a time (1868) when Pasteur's studies on fermentation and putrefaction had produced an atmosphere favourable to the idea that inflammation and pus formation in wounds might also be due to the activity of bacteria. Carbolic acid was perhaps not the ideal disinfectant to use, but once the idea of preventing entry of bacteria into wounds had been grasped by surgeons the way was open for a progressive development of aseptic technique. Concomitantly there was a gradual recognition of the sources from which infection was likely to enter wounds and of the means of dealing with the various dangers.

The idea of specific chemical destruction of bacterial or other parasitic microorganisms without damage to the infected host was probably first formulated by Ehrlich. It arose in embryo in his very first contribution to science at the age of twenty-three. This was concerned with the use of synthetic dyes in staining histological preparations and blood cells and laid the basis of almost all subsequent staining methods. The differential staining of different parts of cells gave rise to a conception which later became Ehrlich's side chain hypothesis upon which his immunological theories were based. His view was broadly that the various functions of living matter were mediated through chemically distinct "side chains" which reacted appropriately with physiological substances and according to their nature with drugs, toxins *et cetera*. If there were vitally important side chains on the protein of the parasite which were not represented in the host they might allow an effective chemotherapeutic attack on the parasite *in vivo*. Up to the present all the effective chemotherapeutic agents have been found by what were essentially empirical methods, but the stimulus to search for them has been in part the belief from theoretical grounds like those of Ehrlich that they must exist *plus* the knowledge that at least one natural product, quinine, did exist with something of the required properties.

There is no need for me to do more than mention the amazing success of penicillin and its followers, a success that has changed the whole aspect of medicine and of medical bacteriology.

4. Pathogenic Organisms are Highly Variable in their Characteristics Including Virulence for any Given Host. To a Large Extent this Variability Can Be Controlled in the Laboratory.

The variability of microorganisms as agents of disease is probably implicit in the changes in the virulence of certain diseases from time to time. In 1858 for instance diphtheria rather suddenly took on the classical form and spread rapidly around the world. Previously it had been seen much more as laryngeal croup than in the faecal form. In the autumn of 1918 pandemic influenza took on a highly lethal character that must have represented a mutation of the virus.

The importance of microorganismal variation for medicine was, however, first recognized by Pasteur in experiments with the bacillus of fowl cholera in 1880. If virulent cultures were allowed to age and were transplanted to fresh medium only at intervals of some months they became progressively less virulent for chickens. The birds which had received the attenuated culture were, however, then fully resistant to inoculation with a virulent culture. Pasteur recognized the similarity of the process to Jenner's

vaccination against smallpox with cowpox "matter", and for the rest of his life concentrated on the development of methods of vaccination against infectious diseases by the use of attenuated living microorganisms. In the light of modern knowledge Pasteur's work is by no means as impressive as it was to his contemporaries, and even yet the processes by which a microorganism can be made to lose virulence yet retain immunizing power are by no means fully understood.

Most of the present-day applications of the Pasteurian method are in veterinary diseases, but our sheet-anchor of protection against the only two killing diseases against which we have no effective chemotherapy is still immunization with attenuated virus. Most virologists now believe that the vaccinia virus used in Jennerian vaccination is not derived from cowpox, but is an attenuated strain of smallpox virus. Immunization against yellow fever is with the variant 17D administered as living virus.

Immunization with killed microorganisms or products of their action is an extension of Pasteur's ideas, due largely in the first instance to Almroth Wright.

5. A Pathogenic Organism Does Not Always Cause Disease—Subclinical Infection is Extremely Common and Frequently Immunizes the Subject against Subsequent Serious Infection Due to the Same Species of Organism. This Natural Immunization Can Often be Imitated by Artificial Means.

The idea of subclinical infection basic to the present-day understanding of such diseases as diphtheria, meningococcal meningitis and poliomyelitis was one of the hardest for early bacteriologists to grasp. The only comprehensive history of infectious disease in Britain was written by Creighton between 1884 and 1890. It is a book of great learning and of astute understanding, but in it Creighton dismisses the germ theory of disease as untenable. It was probably the crudity of the early bacteriologists' conceptions of the relation between bacteria and disease that repelled Creighton's subtle intellect. Until bacteriologists felt they could admit in public that the bacteria they had shown to be the cause of this or that disease could exist in the bodies of many healthy people without causing symptoms, men like Creighton and Bernard Shaw could continue to scoff at the germ theory of disease.

The concept of subclinical infection first became really clear as a result of studies on diphtheria, particularly those of Park and Zingher in the 1920's. In New York at that time only a small percentage of children suffered an attack of clinical diphtheria during childhood, but the vast majority showed a change from the Schick-positive to the Schick-negative state by the time they were fourteen. It appeared that the normal sequence was for children to be infected in the throat by diphtheria bacilli which for one reason or another (small dose or low intrinsic toxicity, for example) produced small amounts of toxin to act as an immunizing stimulus but no symptoms. Symptomatic infection gradually came to be regarded as the unusual condition that resulted when a child had the misfortune to be infected with a large or unusually virulent dose of infection before he had had the opportunity of acquiring an immunizing subclinical infection.

Recent work on poliomyelitis has indicated that the situation is very similar in principle to that existing in regard to diphtheria. Here there is an interesting additional feature that the earlier the age at which first infection occurs, the less likely is paralytic invasion of the central nervous system likely to eventuate.

The most rational way to look at artificial immunization against any of the common endemic diseases is to regard it as a substitute for the uncertain process of natural subclinical infection. No type of immunization provides complete protection, but after the use of the appropriate immunizing agent we can feel sure that a child has received a safe antigenic stimulus and is no longer exposed to the chance of receiving his first infection with a virulent disease-producing dose.

The Control of Infectious Disease.

To some extent each of these principles is applicable to every infectious disease, although individual circumstances will determine which of them is most important in the practical problems of control. Many variables have influenced the speed and effectiveness with which control has been obtained. Yellow fever of the tropical ports of the West Indies and Central America was speedily eliminated as soon as it was clear that *Aedes aegypti* was the sole vector. It is a domestic-breeding mosquito, particularly easy to control. Poliomyelitis, on the other hand, has, despite an almost unlimited expenditure of money, offered no prospect of control until very recently. The difficulties here have been for the most part related to the technical difficulty and expense of experimental work on the virus and on the other to the actual character of the human disease.

In considering the influence of applied microbiology on human affairs we must be careful to remember that "effective applied microbiology" is much older than the science of microbiology. By far the best preventive of infectious disease is the development of a social pattern in which cleanliness and good manners become possible with an adequate standard of living. It is interesting to note how the rising standard of living has always been associated with increased care in the handling of all types of potentially infectious human secretions. "Good manners" are nearly always opposed to the spread of infection. There were great public health protagonists before bacteriology. Captain Cook's insistence on antiscorbutic food and cleanliness below decks was as effective in his ships as Florence Nightingale's common-sense measures were in the barracks hospital at Scutari.

There will be little serious infectious disease in any community where living standards are such that excreta are efficiently disposed of, mouth and nose secretions handled according to the social code, and biting insects denied access to human beings.

The successes of preventive medicine have been largely, though of course not entirely, in the prevention of disease in environments highly favourable to its spread. War notoriously has always favoured infectious disease of every sort and the greatest triumphs of applied microbiology have been in removing the dangers of disease from military operations.

I do not want to present a lot of graphs and figures showing the reduction of general and of infantile mortality over the last hundred years. You are all aware of the steady increase in the expectation of life at birth that has taken place and of how the mortality from virtually all of the infectious diseases has fallen year by year. What I would stress is that to a very large extent this improvement has taken place quite irrespective of specifically medical measures. The death rate in New Zealand in 1876 was only 11.6 per thousand at a time when bacteriology had not yet become a science. Tuberculosis rates were falling steadily long before any specific measures had been initiated to control its incidence.

We have to look at the whole process of developing civilization to understand its influence on infectious disease. In the more advanced western communities the process has been essentially one of overcoming the biological disadvantages of life in industrialized urban communities. Perhaps the most important phase has been to provide the equivalent of space in crowded surroundings. Sanitation on the one hand and the organization of safe supplies of water and food on the other fall into this category. Men in the food-gathering or nomadic pastoral stages of human development are not going to have many opportunities to pass intestinal bacteria to one another, however careless they are in their habits.

In countries where insect-borne diseases are or have been important, notably malaria, indirect amelioration of the conditions by the draining of marshes is often very important, but in general specifically directed measures are required. It is very interesting to see how wide a variety of control measures were needed to complete the eradication of malaria from the southern United States within the last

ten years. It was a matter of bringing all the resources of a scientific civilization to bear on the ecological, social and medical conditions relevant to the spread of malaria. Yellow fever control in the tropics has met the same types of problem and solved them by an equal diversity of methods.

Sterilization of infected materials has played a significant part in the improvement, most of all of course in making hospitals places where lives could be saved instead of being centres for the dissemination of wound and puerperal infections. Even in this field, however, hot water and soap provide an ancient technique that was highly effective long before the days of bacteriology.

At a more specifically scientific level we have the use of immunization to protect individuals against infections which they must inevitably encounter. In normal communities only two procedures have been sufficiently widely applied to have had any significant influence on mortality—Jennerian vaccination against smallpox and immunization against diphtheria. In the near future I expect that an effective method of vaccination against poliomyelitis may be developed, but it is not likely that any of the other diseases of civilized areas will be dealt with in this way. I have an open mind about B.C.G. vaccination against tuberculosis. On the whole its use seems called for only as a temporary measure at certain stages in the process of eradicating tuberculosis. In the tropics yellow fever vaccination is of great importance in all areas where jungle yellow fever is endemic. Immunization campaigns against typhoid, cholera and plague are again temporary relatively unsatisfactory measures which will be progressively replaced by the measures which have eliminated these diseases from western civilization.

Finally, the great advance of our own time has been the therapeutic conquest of bacterial, protozoal and rickettsial infections by specific drugs. It is hardly too much to say that at the present time no person previously healthy suffering from any such infection need die if he can be brought to a well equipped hospital before irreparable damage is done to his tissues. Even with the virus infections, chemotherapy of bacterial complications will in most of the common types remove the risk of death.

At this point it would be interesting, I think, to take you back again to 1852 and read what Farr wrote in his report on the census of 1851:

... The prolongation of the life of the people must become an essential part of family, municipal and national policy. ... Science indeed can scarcely apprehend all the results and all the modifications of society that would flow from the extension of life to its natural limit nor perceive how all the violence, impurity, ignorance and innumerable diseases which now destroy men, can be dispersed. But science offers no justification for despair.

Within one hundred years we have been able to disperse some of that violence, much of the ignorance and virtually all of the innumerable diseases to which Farr refers. And we begin to see some of his "results and modifications of society".

In 1952 we can look upon infectious disease as conquered. All but two of the great killing pestilences of the past are susceptible to effective drug therapy and against these two—smallpox and yellow fever—highly effective immunization is available. Those virus diseases against which neither immunization nor therapy has been developed are for the most part trivial (measles, mumps *et cetera*) or rare (for example, encephalitis) conditions. The most conspicuous failure is poliomyelitis, but as I indicated I feel very confident that in a few years' time an effective means of immunization will have been found. We can never feel quite confident that there are no unpleasant surprises in store for us amongst the virus diseases. There are three famous epidemics of the past which I believe to have been virus diseases and which will serve to indicate the possibilities of the unexpected. These were the English plagues of the Tudor period, the 1918-1919 pandemic of influenza and the low-grade pandemic of *encephalitis lethargica* which reached a peak in 1923 and disappeared about 1930.

In Western countries we have reached the stage that every child that survives the neo-natal period can expect to live sixty years; accident of one sort or another will of course intervene in a small proportion. This is a situation quite unique in the whole history not only of man but of life itself. Never before has there existed a living species 90% of whose offspring survived to reproductive age. There is no physical or biological reason why this should not hold for all races and communities throughout the world. The only obstacles are social and economic. We know what is needed, the physical requirements are available and only human effort and the intelligent adaptation of known principles to new situations are required to bring the East to the same level of survival.

In the past, infectious disease, particularly infectious disease of infancy and childhood, was the main agent in keeping human populations at a level with a tolerable relationship to food supply and other natural resources. Today that check is actually or potentially absent.

Irrespective of whether the abolition of infectious disease has been mainly due to deliberate scientific effort or to social and economic processes, its disappearance as an effective social factor is inescapable. There are some important biological implications in this change which may well represent the most important social revolution in history.

In the Western democracies the progressive improvement in life expectation has gone on in parallel with increasing economic resources on the one hand and a diminution in the birth rate on the other. This has allowed an over-all maintenance and improvement of living standards, particularly when, as was usual in the last hundred years, there was extensive emigration to underpopulated fertile areas. Now we are being driven forcibly to look more carefully at this process. Any ecologist knows that in a given territory there is only such and such a population of any given species of bird or animal that can be maintained from year to year. Man is a mammalian species that is not exempt from biological necessity. If we postulate an acceptable living standard for all people, and a given level of technological development, it is possible to say with fair precision even now that the world or any special part of it will support a population of only so and so many million human beings. The general impression at the moment is that the optimal population of the world has already been exceeded and that in large areas of Asia the population is close to the maximum that the land can keep alive.

In two countries, Great Britain and Sweden, official commissions on population have accepted as inevitable and desirable that policy in those countries should be towards the maintenance of a stabilized population level. Where there is an expectation of life of sixty to seventy years, this means that by one means or another the birth rate of the stabilized population must be reduced from the "physiological" rate of approximately 50 per thousand down to 14 or 15 per thousand. Each married pair must produce on the average somewhat less than three children to maintain the stable situation. It is very interesting to note how acceptable this level appears to be to the average people of the community. Of my own acquaintance I know hardly a family with less than two or more than four children. Statistics bear out the contention that young couples nowadays waste little time in having two children but then become increasingly averse to further enlargement of the family. It is complete sophistry to look for any other explanation of the facts than deliberate interference with the process of conception in order to conform with the couples' sense of what is acceptable to them and to the community. In all countries of the world it will soon be a short-term biological necessity to attain stabilized population levels, and the experience of all liberal democratic countries is that the necessary pattern of reproductive behaviour will be readily adopted by any community to whom the knowledge and materials for effective family limitation are available. Given an absence of major wars, there is every reason to believe that a provisional solution of this type could be obtained in a reasonably short period.

Unfortunately this is not a satisfactory long-term solution of the problem that we have created by abolishing infective disease. If we ensure in general terms that each pair produce two children, both of whom reach reproductive age and that the process continues indefinitely, we must inevitably come up against the problem of genetic deterioration. Unless the whole of modern genetics and evolutionary theory is wrong, the stable population must in the long run undergo progressive genetic deterioration. The whole structure of the evolutionary process depends on the fact that mutation is constantly occurring and that only by a highly selective winnowing of the various gene-combinations that arise can evolutionary progress or even maintenance of quality occur. The vast majority of gene changes must of necessity be disadvantageous to the carrier. To maintain the normal character of the species every appearance of a deleterious mutation must be counteracted by a corresponding "genetic death". As Muller has pointed out, where a deleterious mutation does not produce death or seriously impede reproduction, the characteristic is liable to spread through succeeding generations like a slow-moving infectious disease. If all haemophiliacs could be rendered healthy by some simple maintenance therapy and if there were no direct interference with their breeding, Muller calculated that in about 50,000 generations half the world's population would suffer from haemophilia.

In the past every human generation passed through a test for survival that eliminated approximately three-quarters of the children conceived. We can feel reasonably certain that this massacre of the innocents did fall significantly more heavily on the genetically inefficient than on the normals. In the absence of selection for reproduction by Nature's method of survival or death, we can look forward to a steady deterioration unless some effective alternative method of selection can be devised and implemented. It must be remembered too that if circumstances are such that the genetically inefficient should have a higher reproductive rate than the genetically sound, deterioration will proceed proportionately more rapidly. All studies of the inheritance of intelligence seem to agree that there is a negative correlation between the intelligence of children and the number of siblings in the family. Burt has calculated that the intelligence quotient of London's population is falling from this cause about 1.4 points per generation.

I know that it is not popular to talk about such matters and that it is still "correct" for orthodox geneticists to state that we know too little about human genetics to be sure even that this process of genetic deterioration is going on, let alone to devise any means of slowing it down or reversing it. Nevertheless, I feel very strongly that it is a reality and that it presents by far the most important human problem that confronts us. Modern theory of evolution and population genetics is well advanced and accepted in every part of the world except where the blight of Lysenkoism is upheld by Soviet totalitarianism. Selective survival is still the essence of evolutionary progress. There is a striking phrase of R. A. Fisher's about the miracle of evolutionary advance made possible by selection "in the teeth of a storm of deleterious mutations". If we accept the origin of the human species by natural evolutionary processes, there is no possible escape from my contention that the current ideal of the stable population and the two-child family will in the long run have disastrous results.

This may seem to you very remote from my theme of the impact of microbiology on human affairs, but I would remind you that the situation has arisen directly from the disappearance of infectious disease in childhood as the major agent for the elimination of deleterious gene-combinations. I am proud to have taken some part in the later stages of the development of the science of medical microbiology and its application to human problems. That process which I have briefly described has been the most effectively beneficial of all human activities in history. We have removed the major cause of untimely death and greatly increased the sum of human happiness. The revolution has been virtually completed. Now we must

look at the deeper implications of what we have done and define what are the new problems that medical science must face and overcome.

The conquest of infectious disease was the work of the century with which I have been concerned, 1852-1952. The great problem of medicine from now onwards is the vastly more difficult one of dealing with the genetic aspects of disease and of racial deterioration.

Men entering the field of medical research before 1930 tended to go into medical bacteriology or classical physiology, then came biochemistry and electrophysiology, which are still the fields that attract most recruits. Bacteriology nowadays has an almost wholly biochemical aspect and the modern physiologist needs primarily to be an electronic engineer. It is a pity that so much of modern medical research is so deeply concerned with the minutiae of chemical and physical functioning within the body that there are few men of the highest quality who have any time to look at the broader biological aspects of health and disease. If I had to advise a young medical graduate of outstanding ability as to the post-graduate training that would fit him best for the important coming tasks of medical research, I should tell him to concentrate on genetics. On a background of classical genetics he should build on the one hand a sufficient understanding of biochemical genetics and on the other an interest in population genetics and evolutionary theory. With such a training he could find a world of interest in the problems of human constitution and disease.

Similarly, if I were asked to advise an individual, an organization or a government as to the best use of a large sum of money for the advancement of medical knowledge, I should have no hesitation whatever in recommending the formation of a comprehensive department of medical genetics in one of the larger medical schools. I do not think it is widely enough realized what potent techniques for the study of human genetics are now available. The twin study method is the most important. If I were in charge of such a department I should try to implement a project that might offer an amazing yield of information. It would be simply to ascertain from every patient entering a major teaching hospital whether he or she had a twin. If so the co-twin would be sought out and persuaded to submit to a medical examination in regard to those aspects relevant to the condition for which the index twin sought advice. Incidentally, it would be ascertained whether the twins were identical or non-identical. By comparison of correlations between identical as against non-identical twins a wealth of information in regard to the constitutional aspects of disease would rapidly be accumulated. The work of Kallman and Reisner on tuberculosis along these lines was to me the most important development in the study of that disease since Koch's isolation of the tubercle bacillus.

Whatever we feel about the present social and political impossibility of any programme of practical eugenics, we can be quite certain that decade by decade the importance of action will be progressively more widely realized. Sooner or later action will have to be taken and it is our responsibility to initiate the process of accumulating valid knowledge on which eventual action can be soundly based. I repeat that this is the most important task of medical research.

I should like to end this lecture by returning to my own direct scientific interests in microbiology. I have been speaking of the importance of the genetic approach to human medicine, but I have said little about that most exciting phase of modern microbiology, the application of genetic methods to microorganisms. Within the last two years it has been shown that a form of hybridization is possible between microorganisms formerly considered to reproduce wholly by asexual methods. This was shown for bacterial viruses (phages) by Luria and Delbrück, for coliform bacilli by Lederberg and Tatum, and for influenza viruses by Lind and myself. A new field has been opened up that is now being actively explored. It is at present a highly academic pursuit as remote from practical affairs as Faraday's experiments on electromagnetic induction or

Rutherford's demonstration of the atomic nucleus were at the time of their announcement.

Perhaps I have too vivid an imagination, but I am profoundly disturbed by the future potentialities of such work. In the course of a hundred years we have banished infectious disease and in the process gained an increasing understanding of the nature of pathogenic microorganisms. Today there is little human need for further knowledge of how bacteria and viruses produce disease; at most such knowledge can help us only a little further onward in our approach to perfection in the control or treatment of infection. Yet there is greater activity in this field than at any previous time. It seems that we may soon reach an understanding of the nature of virulence, of the action of antibiotics and of the significance of serological character in relation to immunity. We may learn ways of controlling the character of microorganisms and of producing combinations of characters that could not have arisen in Nature. Just as in 1939 many physicists hoped in their hearts that further research would show that the fission of U^{235} could not be made the basis of an atomic explosive, so we may hope that the problem of creating unnatural pathogens of terrifying potentialities as weapons of war will prove too difficult for human ingenuity.

Perhaps I can draw together the threads of this Cassandra-like discourse by repeating what thoughtful biologists have been saying for many years—that man is a species of mammal that has arisen by the normal processes of evolution and that he can survive only if he takes due regard of the processes that have brought him into being. His manipulative ability of hand and eye, his intelligence, language and his over-riding demand for power over his environment and over his fellows have led him progressively into a series of ecological traps.

Urban life, a necessity for the development of civilization, inevitably led to the development of infectious disease. I have described how the science of applied microbiology has liberated us from that ecological trap but led us into the other of overpopulation. Birth control can save us from the evils of overpopulation, but confronts us with the new trap of genetic deterioration. Wherever we utilize knowledge for the short-term satisfaction of our desires for comfort, security or power, we are all too prone to find that on the long-term view we are creating one more ecological trap from which sooner or later we must extricate ourselves.

I have no solution to offer except to hope that in one form or another an understanding of and interest in the long-term aspects of human biology can be diffused through the world. Current history is determined by the actions of men trained in economics, in history, in military science or in theology, and self-trained in the technique of obtaining and maintaining power. I doubt if there is a single politician in this country, whether of the right or the left, who has any knowledge of the broader biological approach to human problems. When the necessary biological attitude is developed in those who hold positions of power and authority we can hope for a gradual change to policies based on long-term views.

DISSECTING AORTIC ANEURYSM.

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while a previously baffling confusion of signs and symptoms related to various regions and systems may be readily explicable in relation to a progressive aortic dissection. Most cases can be seen—at least in retrospect—to show characteristic features, which may indeed be virtually pathognomonic when considered in the light of their time-relationship with other events in the illness.

Frequency and Age and Sex Incidence.

The incidence of dissecting aneurysm is greater than is generally appreciated; but owing to the varying degrees of selection of material, published figures vary. Thus in Mote and Carr's (1942) series, gleaned from the San Francisco Coroner's Court, and therefore comprising predominantly cases of sudden death, it figures prominently with a frequency of one in every 100 non-violent cases, while the less acute and more clinically important forms which are seen in general hospitals are quoted as occurring in from one in 500 to one in 300 autopsies. At the Royal Adelaide Hospital there have been approximately 7000 deaths in the last five years, of which 13 have been shown at autopsy (or operation) to have been due to dissecting aneurysm—a frequency of one in 500. Of these cases four were correctly diagnosed *ante mortem*. In a proportion of cases the condition runs a chronic course and usually escapes recognition unless discovered incidentally at autopsy, death commonly having been due to some intercurrent disease. Shennan (1933) described 79 of these so-called "healed" aneurysms among the 300 documented cases he collected; and recently more of these cases have been diagnosed.

Subjects in the fifth and sixth decades are those most commonly affected, and men outnumber women in the proportion of three to one. There is a high incidence of hypertension, and this may explain the appreciable frequency with which dissecting aneurysm complicates coarctation of the aorta and pregnancy when found in the lower age groups. In Adelaide the subjects were all, save one, aged from sixty to seventy years, and 10 of the 14 were men.

Pathogenesis.

The occurrence of dissection appears to be determined by a combination of disease of the aortic media, permitting the ready separation of its layers, with a certain degree of hypertension. The cause of the lesions of the media remains obscure in spite of considerable investigation, and they do not appear to be specific. Erdheim's "cystic mediocarcosis" is merely one variety, and cases have been described in which focal or diffuse changes affected either the fibrous, the muscular, or the elastic tissues of the media. Luetic infection mats together the laminae of the media and renders dissection almost impossible, so that syphilitic aneurysms very rarely dissect. Atheroma, as might be expected in view of the age groups affected, is of common occurrence, but is related probably only incidentally, except in the relatively few cases in which secondary dissection follows partial rupture of an atheromatous weakening of the aortic wall. In Case XII this process was present, and such cases should possibly not be regarded as of true dissecting aneurysm. In dissecting aneurysm cleavage of the media probably precedes rupture of the inner coats, and a small number of cases have been reported (Tyson, 1931; Mote and Carr, 1942) in which extensive dissection has occurred in the absence of any intimal penetration. The space usually contains blood-stained fluid and may result from bleeding of *vasa vasorum* into a degenerate area. In most cases, however, the inner coats are ruptured transversely just above the aortic valve. It is this region which is subjected to the greatest mechanical stress, owing to its receiving the impact of the blood-stream ejected from the ventricle in systole, plus the "water hammer" effect of the blood column rebounding against the closed aortic valves and producing a considerable force which is directed longitudinally at first; then, as the "water hammer" is brought to a halt, circumferentially. Thus the ascending aorta is subject to an alternation of extension forces and bursting pressures. In syphilitic aortitis the elastic components of the walls are

FROM being a pathological curiosity, dissecting aneurysm of the aorta has, in less than two decades, been appreciated as producing a variety of clinical pictures which, although conforming to no set pattern, can yet be recognized in a large percentage of cases, provided that clinical suspicion is frequently enough aroused. Bizarre or atypical features in a case of possible myocardial infarction, or of apparent peripheral arterial embolism, or of cerebral vascular accident may provide a pointer to the correct diagnosis;

destroyed and the walls are gradually hammered out to form the characteristic fusiform dilatation. In dissecting aneurysm, on the other hand, it appears that the inner coats lack the support of the outer coats against elongation strains, owing to the absence of the normal anchoring of one to the other, so that the inner coats become disrupted, and blood then bursts through the defect into the cleavage space. The part played by the diastolic recoil in producing the rupture was strongly advocated by Shennan and appears to accord with hydrodynamic laws and explain adequately the common location and character of the rupture. Exertion is a frequent but not invariable precipitating factor, presumably by causing a temporary further increase in intravascular tension.

After rupturing through the intima and inner two-thirds of the media, usually in a linear or T-shaped transverse tear two to three centimetres above the aortic valve, the dissection remains in this plane of the media, extending about more or less of the aortic circumference, and proceeds sometimes rapidly and completely, sometimes more slowly and progressively, sometimes only to a limited degree, along the aorta and its major branches. The plane of cleavage is often very free and one then finds almost complete dissection of the coats extending throughout the length of the aorta and for a considerable distance along the great vessels. Cleland (1951) has stated that this cleavage plane exists in healthy aorta; but a series of measurements of the resistance to separation at various levels of the media in fresh autopsy specimens has not confirmed this (see appendix). Certainly the pressure required to introduce fluid between the layers is such that even severe hypertension is unlikely to cause dissection in the absence of disease of the collagen and elastic strands spanning the medial laminae (Halliday and Robertson, 1948; Robertson and Viner Smith, 1948).

At any stage the adventitia may rupture and pour blood into one of the body cavities. In up to 50% of cases the intrapericardial portion of the ascending aorta is involved with consequent haemopericardium and the concomitants of acute cardiac tamponade. In some 25% the rupture occurs into the left hemithorax, either intrapleurally or extrapleurally, death usually ensuing rapidly; or a slow leak may continue for some days or weeks before death. Rupture may likewise occur retroperitoneally. In the absence of rupture, death may result from the consequences of arterial occlusion or from cardiac failure. Particularly when reentrant rupture into the aortic lumen occurs or when the dissection remains intramural without intimal tearing, the patient may survive these hazards; and it has been stated (Weiss, 1935) that 10% of the patients recover. The survival rate may be higher, as indicated by Shennan's figures (79 "healed" cases in a total of 300), and the series reported by Glendy *et alii* (1937), in which in six cases out of 19 the condition was found incidentally after death from other causes.

Symptoms.

The clinical picture, which may be divided into initial, intermediate and terminal stages, can be appreciated only in relation to the pathological process, and will be considered accordingly.

The initial rupture produces violent nervous stimulation, with usually agonizing substernal pain, pronounced neurogenic shock and often syncope. The pain at first often radiates up into the neck, but rarely into the arms, a point which may assist in the differential diagnosis from myocardial infarction. Further progress of the dissection produces a recurrence of the pain, of greater or less severity, which may be felt successively in the arms, back, epigastrium and legs. This picture by no means always obtains, and there may be a curious absence of pain throughout, or the pain may be transient or of only slight severity. In only half of Baer and Goldburg's (1948) series was pain sufficiently severe to be recorded, while in the Royal Adelaide Hospital cases one patient had no pain and in two others it was very transient. This stage often mimics myocardial infarction, but may be differentiated from it by the frequent maintenance of blood pressure after recovery from the initial shock, by the electrocardio-

gram, which usually shows the pattern associated with hypertensive left ventricular "strain", and by associated atypical features such as pulse differences *et cetera*.

After this sometimes unobtrusive initial phase there usually develop symptoms due to the occlusion of the aortic branches. The mechanical basis for the occlusion may be collapse of the lumen due to infolding of the inner coats, shearing off of smaller branches by the dissecting mass, or rupture of the intima and thrombosis of the vessel (see Figure I). However, an artery shown at

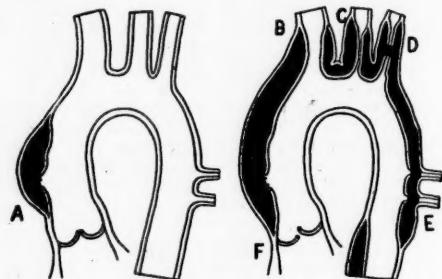


FIGURE I.

Showing some mechanisms producing arterial obstruction in dissecting aneurysm. (Modified in part from Halliday and Robertson, 1946.) A, site of intimal rupture; B and C, occlusions respectively partial and complete, due to inward bulging of the intima; D, the same, with superadded rupture of the inner coats; E, "shearing off" of smaller vessels such as the intercostals; F, possible mechanism of the aortic incompetence complicating supravalvular dissection.

autopsy to be extensively involved by circumferential dissection frequently appears to have functioned quite well during life, while large arterial branch similarly involved may be at first completely occluded, the result being complete cessation of arterial supply to a limb, with later reestablishment of an adequate circulation. This may be due to the collateral supply, but it seems likely in many cases that a considerable temporary arterial spasm is also present. The presenting picture now depends on the extent and distribution of the occlusive process; when the trunk vessels of the limbs are involved, the condition may simulate subclavian or femoral arterial embolism or saddle embolus of the aortic bifurcation if the occlusion is complete, while narrowing of one subclavian artery may be recognized only by detection of pulse or blood pressure differences between the right and left sides. When abdominal visceral branches are affected there may be pain simulating peptic ulcer, together with haematemesis and melena, or the picture of mesenteric thrombosis with intestinal obstruction may appear. Renal involvement with haematuria is comparatively rare.

Considerable attention has been drawn to the neurological symptoms, which are very common, and may be divided (Weisman and Adams, 1944) into those due to ischaemic necrosis of the peripheral nerves, the spinal cord and the brain respectively. It may be emphasized, however, that nervous disturbances may be due to temporary ischaemia without necrosis, apparent full recovery taking place after some hours. Peripheral neuropathy is common, and forms portion of the text-book picture of embolism (pain, paralysis and paresthesia), and need not be further considered. Spinal cord ischaemia is common, and frequently produces curiously transient paraparesis with or without sphincter disturbances. In other cases, in which there is complete occlusion of a number of segmental (thoracic or lumbar) aortic branches, from the "shearing off" mechanism illustrated, infarction of the cord occurs, with symptoms of an acute transverse lesion, which may be total; or it may be partial, affecting predominantly motor function and appreciation of pain and temperature. This sparing of light touch and postural and vibration sense is due to the better developed anastomotic arterial chains

posteriorly, and the picture is then identical with that due to thrombosis of the anterior spinal artery. Some degree of ischaemia of the brain likewise is common and may be shown only by mental confusion or aphasia. When complete occlusion of one carotid is allied with a poorly developed or atheromatous circle of Willis, so-called "carotid hemiplegia" with homolateral blindness and contralateral hemiplegia and hemianesthesia will result. As well as ischaemic neuropathy there may be pressure on the recurrent laryngeal nerve, as in the classical descriptions of syphilitic aneurysm, producing hoarseness due to laryngeal palsy. Other pressure symptoms are not often seen, but include dysphagia and a constant dull pain in the mid-line of the back. Their occurrence usually indicates partial external rupture of the aneurysm.

Occlusion of the coronary ostia is recorded (Glendy, Castleman and White, 1937), but is rare. However, proximal extension of the dissection to the aortic wall immediately above the valve ring does frequently produce an aortic diastolic bruit often with aortic incompetence. This is a most important diagnostic sign, and when appearing in a case of apparent myocardial infarction is almost pathognomonic. When such a case becomes chronic it closely mimics syphilitic aortitis with aneurysmal dilatation and regurgitation, and it should therefore be suspected if the Wassermann test produces a negative result. Gouley and Anderson (1940) reported six of these cases in which the subjects presented with heart failure, and pointed out that haemoptysis occurred in five of them—possibly on account of acute relative left ventricular failure. The exact mechanism of the valvular incompetence is not established; it is certainly not due to any lesion of the valve cusps, but presumably is due to a mechanical disturbance in their points of fixation, with or without dilatation of the aortic ring. That the diastolic bruit is, in fact, frequently evidence of regurgitation is indicated by the coincidence of Corrigan's pulse and Duroziez's murmur in many cases; but in some a less typical murmur may be heard in diastole, which has been said to be due to the flow of blood between the true and the false aortic lumina. In the majority of chronic cases the subjects pass into a stage of severe cardiac failure, associated with a degree of cardiac enlargement greater than one would expect from the hypertension and regurgitation present—indeed, the hypertrophy may be considerable when both these factors are absent or minimal. It is tempting to postulate that the primary factor causing aortic medial disease can also have a deleterious direct effect on the myocardium. The not infrequent development of a pericardial friction rub, and the post-mortem demonstration of pericarditis in many cases may favour this possibility.

Rupture of the external coats constitutes the third phase and is of course almost always a terminal event. As was stated earlier, half the patients die with cardiac tamponade due to intrapericardial rupture; in another 25% bleeding into a pleural cavity occurs, and others have intraperitoneal or retroperitoneal extravasation. Although the burst is usually of catastrophic magnitude, there may be only a slow leakage of blood, especially into a pleural cavity, so that an originally "sympathetic" serous effusion may become first lightly, then heavily blood-stained. Should the pleura remain intact, an effusion may be mimicked by a large extrapleural haematoma. Patients who survive for several weeks or longer most commonly die finally from external rupture; but in a number rupture does not occur and death may result from other effects of the aneurysm, such as congestive cardiac failure or cerebral thrombosis, or from some unrelated and incidental condition.

Reports of Cases.

Cases I, II, III and IV illustrate the common clinical picture mimicking myocardial infarction.

CASE I.—A previously healthy business man, aged sixty-seven years, was driving his motor-car when he felt a sudden severe pain in his chest. He stopped the car and after about three minutes the pain ceased, but left him feeling too weak to move, and he vomited once or twice. He then collapsed and was brought into hospital, when he was seen to be pale and shocked, with a systolic blood pressure of 75 milli-

metres of mercury and a diastolic pressure of 50 millimetres. The heart sounds were soft and regular—no bruits were heard. Some upper abdominal tenderness and muscle guarding were present. He was thought to have sustained a myocardial infarction, but his electrocardiogram revealed no abnormalities. He vomited a good deal for twenty-four hours after his admission to hospital, and his abdomen remained tender, but he complained of very little pain in his chest. Three days after his admission to hospital a diastolic bruit was heard at the base of the heart, the blood pressure then being 150 millimetres of mercury, systolic, and 75 millimetres, diastolic, and a diagnostic Kline test was found to produce a negative result. The patient seemed to be improving when he suddenly died, six days after his admission to hospital.

At autopsy a T-shaped intimal tear was found just above the aortic valves, whence dissection had extended almost to the aortic bifurcation. The aneurysm had ruptured into the pericardium, which contained about a pint of blood.

CASE II.—A housewife, aged forty-nine years, healthy except for "high blood pressure—210—for years", suddenly developed a severe pain in the upper part of her left arm, which in a few seconds radiated up into the left side of the neck and into the precordium. She was put to bed and apparently given some morphine orally, without much result. She was admitted to hospital thirty hours later, when her extremities were noted to be rather pale and dusky, and her blood pressure 110 millimetres of mercury, systolic, and 80 millimetres, diastolic. Her heart rate was 110 per minute and an aortic diastolic bruit was clearly heard. The lungs were clear on clinical examination and no abnormality was discovered in the abdomen. There was slight oedema of the ankles, and Babinski's sign was present on the right side. The electrocardiogram showed a prominent *Q* wave in lead III with inverted *T* waves in leads II and III, and was considered as being consistent with a posterior infarct; however, the significance of the diastolic bruit was appreciated, and the correct diagnosis was made.

Two days after her admission to hospital the patient became irrational, and the electrocardiogram revealed multiple ventricular extrasystoles. She remained mentally confused and her general condition progressively deteriorated. Atrial fibrillation commenced after a further two days, and she died suddenly six days after the onset.

Autopsy revealed a horizontal intimal tear above the valves, extending around about two-thirds of the aortic circumference, with dissection extending some three inches along the ascending aorta. Rupture had occurred into the pericardium and the right pleural cavity, each of which contained about 25 ounces of blood.

CASE III.—A railway worker, aged sixty-one years, was having his evening meal when he felt a sudden pain in the middle of his chest and in the epigastrium, which was "severe enough to make him perspire". Immediately afterwards he "lost the use of his legs", but about an hour later he found he could move them quite normally again. He was immediately brought into hospital, looking pale but not showing any acute distress. His blood pressure was 105 millimetres of mercury, systolic, and 30 millimetres, diastolic, and the heart sounds were soft and regular, with no bruits. Examination revealed no other abnormalities, save some epigastric tenderness. The reflexes were not recorded. On the following day a low rumbling aortic diastolic bruit was heard, and it was noticed that his pulses felt different. On the right side his blood pressure was 150 millimetres of mercury, systolic, and 110 millimetres, diastolic, whereas on the left it was 190 millimetres of mercury, systolic, and 160 millimetres, diastolic. In the electrocardiogram a rather prominent *Q* wave was present in lead III, and the *T* wave in lead III was at first of low amplitude and later diphasic. There was a leucocytosis of 18,000 cells per cubic millimetre. Three days after his admission to hospital the patient died suddenly.

At autopsy a transverse intimal tear one inch long was found one and a half inches above the aortic valve. The media had been split down to the valve and up into the innominate artery and both carotids to their bifurcations, as well as down the length of the aorta and portion of the left common iliac artery. Rupture had occurred into the pericardium. Very little atheroma was found in the aorta.

CASE IV.—A man, aged sixty-eight years, was examined ten days after he had had an attack of pain in the front of his chest while arising from his armchair. The pain radiated to his neck on the left side and into his left arm, and had persisted ever since. He felt breathless at the time, and breathing tended to exacerbate the pain.

On his admission to hospital his blood pressure was 175 millimetres of mercury, systolic, and 100 millimetres,

diastolic; his heart was not enlarged on clinical examination, and no bruits were heard, but an apical triple rhythm was present. Percussion demonstrated impaired resonance over the lower zone of the left lung posteriorly, together with diminished breath sounds and some tracheal deviation to the right, suggesting a left-sided effusion. An X-ray film of the chest revealed an extensive opacity of the left lung field with its denser portion situated below the clavicle. The electrocardiogram was normal.

Nine days after his admission to hospital the patient had a large haemoptysis and died.

At autopsy examination of the aorta revealed a moderate degree of atheroma. The intimal tear occurred at the summit of the arch and dissection had extended down to the origin of the celiac artery. The adventitia had ruptured near the intimal tear, and a large haematoma had burrowed from the superior mediastinum into the upper part of the left hemithorax retrosternally. From here secondary rupture into the pleural space had apparently occurred, and an encysted mass of clot was found in the lower part of the space posteriorly. A hemorrhagic area in the gastric mucosa was seen at the *fundus ventriculi*, and the brain was said to be "unusually soft".

Cases V and VI both presented as cerebro-vascular accidents, while in Case VII the patient was thought, on his second admission to hospital, also to have had a cerebral thrombosis.

Case V.—A woman, aged sixty-three years, was found unconscious in her home and brought into hospital. She was there found to be in a light plane of unconsciousness, though she was moving her right arm and right leg. Her pupils were equal, both eyes deviated to the right, and there appeared to be left facial paralysis. The left arm was motionless and flaccid, while the left leg was somewhat spastic. The tendon reflexes of both lower limbs were hyperactive, as were those of the right upper limb, but no reflexes could be elicited from the left arm. Bilateral Babinski reactions were present. The pulse rate was 80 per minute and the blood pressure was 150 millimetres of mercury, systolic, and 70 millimetres, diastolic; the heart sounds were faint and regular, with no bruits. In the abdomen no abnormality was detected. One hour later the right pupil became semi-dilated and non-reactive to light, and the pulse rate fell to 48 per minute. Ten hours later the patient's general condition appeared unchanged, but now a systolic cardiac bruit was heard. Left-sided hemiplegia was still present, but the reflexes had returned to the left arm and the right plantar reflex was then normal. An electrocardiogram was taken and revealed slight depression of the S-T segment in leads I and II, with inversion of the T waves in leads I, II and IV_r, while lumbar puncture produced clear fluid under normal pressure. No further change in the patient's condition was noted until fifty hours after her admission to hospital; she then suddenly "became very distressed" and died two hours later.

Autopsy revealed an intimal rupture just above the aortic valve, with dissection of its coats extending into the abdominal aorta. There was some atheroma of the aorta and gross atheroma of the circle of Willis with thrombosis of the right lenticulo-striate artery. The left kidney contained an infarcted area. External rupture had again involved the intraperitoneal aorta with resulting haemoperitoneum.

Case VI.—A foundry worker, aged sixty-five years, was apparently well when he suddenly "lost the use of his left arm and leg", became dazed, and vomited twice. When brought into hospital two and a half hours later he was mentally confused and looked pale and cyanosed. His pupils were equal and reacted to light, and paralysis of the left external rectus was present. He had a left facial palsy of the upper motor neuron type, and spastic paralysis of his left upper and lower limbs. As well complete left hemianesthesia was present. All tendon reflexes were hyperactive and Babinski's sign was present on the left side. His blood pressure was 60 millimetres of mercury, systolic, and 30 millimetres, diastolic, and the heart was not apparently enlarged; the heart sounds were very soft. From his son a history was obtained of some shortness of breath on exertion for several years, together with occasional alleged "fits".

Three hours after his admission to hospital the patient had virtually complete return of normal movement and sensation on the affected side, and was mentally much more lucid. He complained of some headache, but no other pain. He appeared to be in fair condition till two days later, when his temperature and pulse rose, and he appeared pale and sweated, but still complained of no pain. His blood pressure was now 110 millimetres of mercury, systolic, and 95 milli-

metres, diastolic, and the apex beat was felt in the anterior left axillary line, while on auscultation a loud pericardial friction rub could be heard. The electrocardiogram showed a biphasic T wave in leads I, II and IV_r, but no other abnormality; a leucocytosis of 11,500 cells per cubic millimetre was present. On the following day the friction rub was inaudible, but now an aortic diastolic bruit presented itself and the blood pressure had risen to 170 millimetres of mercury, systolic, and 110 millimetres, diastolic. The patient's condition remained unchanged, as did further serial electrocardiograms. Five days after his admission to hospital he died suddenly.

Death had resulted once more from haemoperitoneum. There was an internal tear in the ascending aorta, and dissection had involved the length of the aorta down to its bifurcation, and extended up the right carotid arteries. Examination of the heart revealed gross left ventricular hypertrophy, and its valves appeared normal. The brain was normal on macroscopic examination.

Case VII.—A man, aged sixty-seven years, gave a history of having developed shortness of breath and pains in his chest while chopping wood one month before his admission to hospital. The pain was referred to the front of the chest and had been severe at first; but after rest in bed for a week it had eased and now it developed only after exertion. Eight years previously he had had a haematemesis following a period of epigastric pain after meals, and an "ulcer diet" had been instituted; since then he had been well and had had no recurrence of epigastric pain.

On examination of the patient, his heart was clinically grossly enlarged, the apex beat being palpable lateral to the anterior axillary line. The heart sounds were irregularly irregular and no bruits were audible, the blood pressure being 180 millimetres of mercury, systolic, and 110 millimetres, diastolic. No dependent oedema, no venous engorgement, and no evidence of pulmonary congestion were present. The electrocardiogram showed auricular fibrillation to be present, with some depression of the S-T segment in lead I and elevation of the S-T segment in lead III, while gross cardiac enlargement was evident in the X-ray film of the chest. The Wassermann test produced a negative result.

Three days later a pericardial friction rub was heard at the apex, and this persisted for several days. Serial electrocardiograms revealed little change, and examination after a barium meal demonstrated no evidence of gastric or duodenal ulceration. The patient was given 0.75 milligramme of "Digoxin" daily and discharged from hospital to convalesce.

He was apparently fairly well until six months later, when one day he complained of abdominal pain and vomited several times. He became irrational, and when brought to hospital two days later was stuporose and appeared somewhat spastic on the right side, a Babinski reflex being present on that side. Death occurred one and a half days after his admission to hospital.

Autopsy revealed a rupture of the intima one and a half inches above the aortic cusps, with dissection of the aortic coats up to the summit of the arch. Gross left ventricular hypertrophy and adherent pericardium were present. The coronary orifices were patent and the external coats of the aneurysm had not ruptured. Softening of the brain tissue had occurred in the region of the left internal capsule and cerebellum.

Cases VIII, IX and X showed peripheral arterial obstruction as a prominent part of the clinical picture. Case VIII in particular illustrates many characteristic diagnostic features.

Case VIII.—A labourer, aged sixty-seven years, was admitted to hospital complaining of loss of movement and sensation of his right leg for a period of three days. One week before his admission to hospital he had developed coryza, with a sore throat, cough and sputum. He had retired to bed and had then had two or three attacks of fairly severe precordial pain with dyspnoea. At that time he was examined by his local doctor, who found that his blood pressure was 170 millimetres of mercury, systolic, and 130 millimetres, diastolic, with irregular heart sounds and no bruits. The following day his blood pressure remained the same and he seemed better; but three days later, while he was sitting in bed, the pain in his chest returned and then passed into the "small of his back" and down both legs, which became stiff, and he was unable to move them, nor could he pass urine or feces. His doctor examined him two hours after this incident and found flaccid paralysis and complete anaesthesia of both lower limbs. He was given morphine with little relief; and when he was examined again eight hours later his left leg had completely recovered motor

and sensory function, was painless, and had normal tendon and plantar reflexes. The right leg remained as before and the patient's bladder required catheterization.

On his admission to hospital two and a half days after the onset of the paralysis he was seen to be in poor general condition and mentally rather confused. His blood pressure was now 110 millimetres of mercury, systolic, and 60 millimetres, diastolic, and his heart appeared to be slightly enlarged. On auscultation a loud to-and-fro murmur was heard over the precordium, maximal at the aortic area. Both radial pulses were present and of equal volume, and collapsing in nature. The lungs were clinically clear. There was no abdominal tenderness, but the skin in the right lower abdominal quadrant was bluish and almost anaesthetic, and appeared to bulge more than on the opposite side. In the left lower limb normal movement, sensation and reflexes were present, and there was a prominent left femoral pulse, over which a Duroziez's murmur could be heard. On the right side no femoral pulse was palpable, and the limb was cold, bluish, motionless and anaesthetic, with areas of incipient gangrene over the outer aspect of the foot (where the patient had placed a hot-water bottle) and over the buttock. Dissecting aneurysm was diagnosed on the basis of the typical history and findings. An electrocardiogram showed some extrasystoles and an inverted *T* wave in lead III, but no other abnormalities, and the Wassermann test produced a negative result. On microscopic examination the urine was normal.

Three days later he showed some general improvement, and except for the previously described area the circulation to the right lower limb appeared quite good, although the femoral pulse did not return. The flaccid paralysis persisted and there was complete anaesthesia of the limb and of the trunk below the eleventh thoracic vertebra on the right side. Light touch sensation was impaired up to the level of the fourth thoracic vertebra on the same side.

The patient had a fair amount of pain in his abdomen and right thigh and was frequently mentally confused, but his condition remained more or less stationary until eleven days after his admission to hospital, when he became very distressed, with a feeble irregular pulse, pronounced cervical venous engorgement, and Cheyne-Stokes breathing; he died twenty hours later.

At autopsy the heart was moderately enlarged and the serous pericardium had a finely granular appearance. There was a transverse tear of the intima and portion of the media of the aorta immediately above the left and right posterior aortic cusps, dissection having proceeded to involve the ascending aorta about two-thirds of its circumference, the arch and the whole of the thoracic and abdominal aorta, with extension for some distance along the right common and external iliacs, the left common carotid and left subclavian arteries. The aortic cusps themselves appeared normal and the coronary ostia were patent. External rupture had not occurred, but there were two pints of slightly blood-stained fluid in the left pleural cavity.

CASE IX.—A departmental store buyer, aged sixty-three years, was about to get into bed when he developed severe pain in the lower part of his chest. He was examined by his local doctor, who diagnosed coronary occlusion and put him to bed. The pain recurred from time to time, and four days later an electrocardiogram revealed auricular fibrillation. He was given digitalis and quinidine and sinus rhythm returned. On the following day, however, he complained of acute abdominal pain, and five hours later developed severe pain passing down both legs, with numbness below the knees. Both femoral pulses were absent, and he was thought to have had a saddle embolus at the aortic bifurcation and was immediately transferred to hospital.

On examination of the patient he appeared shocked, but his blood pressure was 210 millimetres of mercury, systolic, and 140 millimetres, diastolic. The cardiac apex could not be felt, and the sounds were faint, with a triple rhythm. The abdomen was slightly tender in the mid-line, and aortic pulsation could be felt at the level of the umbilicus. There was still no femoral pulsation, and the legs were cold and white below the middle of the calves, with some impairment of sensation and of movement. An electrocardiogram revealed depression of the *S-T* segment in leads I and IV and inversion of the *T* wave in lead IV, suggesting hypotensive strain rather than infarction, and the diagnosis of dissecting aneurysm was made. However, in view of the less likely possibility that a saddle embolus had lodged at the bifurcation, exploration under spinal anaesthesia was undertaken and the diagnosis was confirmed. Subsequent to operation the patient's condition remained poor and he died twenty-four hours later.

No autopsy was performed.

CASE X.—A motor-truck driver, aged sixty years, while dressing himself one morning, developed a severe pain in the front of his chest as he bent over to put on his shirt. The pain radiated down into the abdomen and he then lost consciousness. When he recovered the pain had eased, but he felt sick and vomited several times, and noticed that his right arm had become numb and that he was deaf in both ears. His previous health had been good, but he had been told by his doctor that he had high blood pressure.

On his admission to hospital he was found to be shocked, greyish cyanosis of his face being present. His right arm was cold and bluish, with a barely perceptible radial pulse, but it could be moved voluntarily quite well. On the left side the pulse was weak, and the blood pressure was 90 millimetres of mercury, systolic, and 50 millimetres, diastolic, no reading being possible on the right side. The heart was not clinically enlarged and the sounds were regular. Examination of the lungs, the abdomen and the lower limbs revealed no abnormality. The electrocardiogram showed slight elevation of the *S-T* segment in leads I and IV, and inversion of the *T* wave in lead III; serial records revealed no significant changes. An X-ray film of the chest revealed cardiac enlargement, affecting predominantly the left ventricle, and the Wassermann test produced a negative result. The condition was diagnosed as a dissecting aneurysm.

On the day after his admission to hospital the patient felt better, but complained of some pain in his chest and of frontal headache. His hearing had improved and the right arm was warmer, but still pulseless. For four days his temperature was elevated to a moderate degree, after which he remained afebrile, and his general condition progressively improved. Only after four weeks was the right radial pulse palpable, and at that time the blood pressure on that side was 70 millimetres of mercury, systolic, and 60 millimetres, diastolic, whereas on the left side it was 120 millimetres of mercury, systolic, and 85 millimetres, diastolic. Otherwise the limb appeared normal and the patient was discharged from hospital.

He was examined at regular intervals during the succeeding eight months, and save for becoming short of breath on exertion and having occasional pains in his chest, he remained well. On the last occasion his blood pressures were as follows: right side, 100 millimetres of mercury, systolic, and 70 millimetres, diastolic; left side, 130 millimetres of mercury, systolic, and 80 millimetres, diastolic.

Cases XI and XII were both diagnosed as acute abdominal conditions, and in the latter case laparotomy was performed.

CASE XI.—A male textile worker, aged sixty-three years, was admitted to hospital four hours after the onset of severe abdominal pain. He had been quite well during the day and had eaten his normal evening meal. The pain was referred to the epigastrium and had developed without warning, causing him to gasp for breath. At the time he felt nauseated and later he vomited once or twice. There had been no relief from the pain since its onset. His health had been fair, but four years previously he had been admitted to the Royal Adelaide Hospital after melena. X-ray examination after a barium enema demonstrated colonic diverticuli, and laparotomy confirmed that finding. However, he had at the time complained of attacks of deep-seated pain in the front of the chest on the right side, to which he had been subject for twelve months. The pain was severe and radiated up into the throat and "nothing relieved it". Examination had revealed no significant abnormality save some cardiac enlargement and a blood pressure of 180 millimetres of mercury, systolic, and 100 millimetres, diastolic.

On the second admission to hospital he was seen to be obviously in pain, and diffuse abdominal tenderness with muscle guarding was present. The blood pressure was now 210 millimetres of mercury, systolic, and 110 millimetres, diastolic, and the heart appeared slightly enlarged; the sounds were regular, with no bruits. His temperature was 96.4° F. and his pulse rate 84 per minute; he was thought to be possibly suffering from pancreatitis or diverticulitis. Seven hours later the patient suddenly became shocked, with dilated pupils, sweating, faint heart sounds, and a fall of blood pressure to 140 millimetres of mercury, systolic, and 70 millimetres, diastolic. An electrocardiogram showed low amplitude of the *T* waves in leads I and IV, but no other change. He had recovered somewhat twenty-four hours later, and then complained of pain in his chest and abdomen, and also of a "stabbing pain" passing up his neck and behind his ears. As well, he found that his voice had become husky. Examination now revealed signs of a left-sided pleural effusion, with dulness to percussion, diminished breath sounds and mediastinal shift to the right. Forty

hours after his admission to hospital he became very shocked and died.

At autopsy the primary internal rupture appeared as a vertical slit 1.7 centimetres in length in the posterior wall of the aorta just distal to the origin of the left subclavian artery. The aorta was dissected along its posterior wall down to the level of the diaphragm, the dissection extending into the right common iliac artery as far as its division. The celiac and superior mesenteric vessels were not involved, but the left renal artery had been dissected for 1.7 centimetres from its origin. The aortic valve was normal and there was some left ventricular hypertrophy. There was extensive hemorrhage into the mediastinum, and the left pleural cavity contained some fluid and one and a half pints of blood clot.

CASE XII.—A man, aged seventy years, was admitted to hospital with a history of severe lower abdominal pain and vomiting beginning two days previously, and both persisting since. After the onset of the pain he had passed several small motions, but had been constipated and passed no flatus for twenty-four hours prior to his admission to hospital. His abdomen was tender and rigid, being most tender a little to the left of the umbilicus. A small left inguinal hernia was present and was somewhat tender. The heart sounds were very faint, and the blood pressure was 70 millimetres of mercury, systolic, and 55 millimetres, diastolic.

A strangulated inguinal hernia was diagnosed and for its relief operation was performed under cyclopropane anaesthesia. The hernial sac was found to contain normal omentum, and a considerable old extravasation of blood was found tracking out through the internal ring, apparently having a retroperitoneal origin. In view of the patient's condition no further exploration was attempted. After the operation his condition seemed fair for a time, but his abdomen became distended and he died two days later.

At autopsy an aneurysm of the abdominal aorta was found, extending from the region of the origin of the celiac artery down to the aortic bifurcation. The external coats had ruptured anteriorly with considerable retroperitoneal haemorrhage.

This case appears to have been due to rupture of a grossly atheromatous area with some secondary dissection, and hence possibly should not be regarded as one of true dissecting aneurysm.

CASE XIII.—Presented as an obscure spinal or intrathoracic condition, and on the recorded clinical findings the correct ante-mortem diagnosis would have been unlikely.

CASE XIII.—A married woman, aged sixty-four years, was admitted to hospital for investigation of pain between the shoulders. She had been well until one month before her admission, when she had developed a continuous "boring" pain in her back at the level of the scapulae. The pain was exacerbated on movement of the trunk, and had been more severe during the week before her admission. On examination of the patient little was discovered; her blood pressure was 185 millimetres of mercury, systolic, and 90 millimetres, diastolic, the heart was not clinically enlarged, and the sounds were regular without bruits. The lung fields were normal. There was tenderness to pressure sharply localized to the spines of the fifth and sixth thoracic vertebrae. No other abnormality was recorded.

An X-ray picture of the thoracic part of the spine showed only slight scoliosis with early osteoarthritic changes, and a blood examination revealed a polymorphonuclear leucocytosis of 12,500 cells per cubic millimetre. The patient's temperature rose to 100° F. every day, but no cause was found for this until a week after her admission to hospital, when she had a rigor, and some dulling of the percussion note and diminished breath sounds were observed at the base of the left lung. A chest X-ray film now demonstrated cardiac enlargement, with displacement of the trachea to the right, and a considerably enlarged aortic knuckle, suggesting an aneurysm. Twelve days after her admission to hospital she died suddenly.

Autopsy revealed a "small rupture" of the aorta opposite the fourth and fifth thoracic vertebrae, with dissection of its coats from the level of origin of the left subclavian artery down to the diaphragm. The sac had secondarily ruptured into the left pleural cavity, which contained about one pint of clot. Examination of the aorta showed extensive atheroma.

CASE XIV.—An example of what may perhaps be termed the "coroner's type".

CASE XIV.—A woman, aged sixty-nine years, was admitted to hospital deeply comatose and was dead on arrival in the ward. Unfortunately no history is available.

At autopsy an intimal and medial tear was found four centimetres above the aortic valves. Dissection had occurred throughout the length of the aorta from this point, involving both common iliac vessels. External rupture had occurred at the commencement of the descending aorta, and there were two pints of blood in the left pleural cavity. Examination of the heart revealed some left ventricular hypertrophy, but otherwise no abnormality.

Summary.

1. Dissecting aneurysm, although uncommon, is an eminently recognizable condition as long as it is considered.

2. It affects predominantly males aged fifty to seventy years.

3. The clinical picture may be readily correlated with the pathological process by considering the following three phases: (i) rupture of the intima and initiation of dissection, with shock, prostration and pain, which may progressively alter its location; (ii) the multiple symptoms and signs due to arterial obstruction; (iii) the final rupture of the aneurysm.

4. Particularly interesting features are the common, frequently transient, neurological manifestations, and the occasional cases mimicking syphilitic aortic regurgitation.

5. Fourteen cases seen recently in the Royal Adelaide Hospital, and presenting most of the usual symptoms, are described.

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Appendix.

Investigation of the Presence of a Free Cleavage Plane in the Aortic Media.

In view of the almost invariable situation of the plane of dissection at the junction of the middle and outer thirds of the media, a number of observations were made in order to determine whether or not a layer existed in this region, in normal aorta, showing significantly less resistance to dissection than other strata; this suggestion had been advanced by Cleland (1951), but doubted by Robertson (personal communication), who had made a number of measurements of the force required to split the aorta in various layers.

Segments of aorta were taken just distal to the origin of the left subclavian artery from twenty subjects undergoing autopsy at the Royal Adelaide Hospital. Prior to autopsy the cadavers had been stored in a refrigerated chamber for a variable period, an average time of about eighteen hours having elapsed after death. The fresh segments were subsequently placed in a sealed jar and refrigerated for a further four to six hours before examination.

After removal of the adventitial coat six longitudinal strips, 3.5 millimetres in width, were cut from each specimen, a

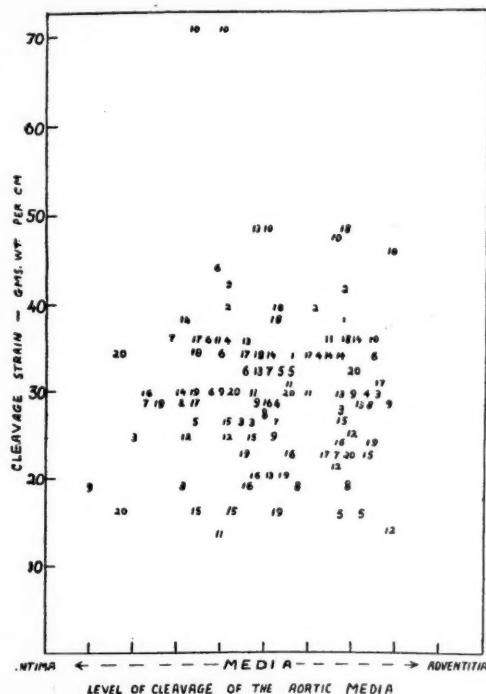


FIGURE II.

Cleavage strains at various levels in the aortic media. Values indicated by the same number relate to the series of measurements obtained from one specimen.

double-bladed cutter being used in order to ensure uniformity, and the media was split for some little distance along them. In each series an attempt was made to place the level of cleavage progressively from intimal to adventitial aspect. The strain required to maintain separation of the layers along the length of the strips by linear traction on the internal and external sections was measured directly by a torsion dynamometer. Subsequently the thickness of each section was measured with a lightly loaded slipping clutch micrometer.

The strain was expressed as grammes weight per centimetre of width of the aortic strip, and the level of the plane of cleavage as a factor produced by dividing the thickness of the internal section by that of the whole of the intimal and medial layers.

When the results are expressed graphically (Figure II) it is seen that the variation between cleavage strains at various levels in any one specimen is considerable, and that between different specimens it is still more pronounced. However, the magnitude of any one set of measurements is comparable and when they are viewed *en masse* it is apparent that at no level of the media is there a significant upward or downward trend in the resistance to separation of the layers.

It is concluded that in normal aorte there exists no level in the media at which resistance to dissection is appreciably lowered. It is possible, and probable, that there may exist such a layer in aorte in which dissections do develop.

PSYCHIATRIC DISORDERS IN GENERAL PRACTICE.¹

By H. M. BIRCH,
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In this talk we thought it advisable to deal with a few everyday aspects of psychiatry as they are encountered in general practice. In doing this we must only touch upon some very profound problems.

But sometimes the ordinary and the elementary need emphasizing, to the exclusion of a lot of airy-fairy metaphysical speculation on one single subject, which leaves you, in the end, with a feeling of "Well, what else could you expect from a psychiatrist?"

I should like first of all to outline the extent of psychiatric disorders in South Australia.

If you consider all the beds in South Australian hospitals other than those devoted exclusively to psychiatry—that is, general, midwifery, tuberculosis and children—the number is roughly 5400. The beds in special hospitals for psychiatric patients total about 3000.

Bearing in mind that many country hospitals have a daily average of beds occupied much below their total bed capacity and that many psychiatric patients are occupying beds in general hospitals, then we may say there are almost as many patients with disturbed mental states occupying beds as there are those with all other types of illness.

What of the psychiatric patients not in hospital?

Only those in general practice can say what percentage of their patients are suffering wholly or predominantly from psychiatric disorders of varying degree.

One consultant physician, who was by no means recognized as having any particular leaning to psychiatry, analysed 500 consecutive patients. He said: "Among the 500 patients there were 116 (23%) without any discoverable organic cause for the symptoms of which they complained. In addition there were 56 (11%) presenting minor organic lesions but with symptoms which could not possibly be explained by the lesions alone. In a word, one-third of the patients suffered solely or predominantly from functional mental disorders."

Nowadays the student in the teaching hospital is surrounded by special branches of medicine and elaborate aids of all kinds from the laboratory, X-ray and other departments, all of which are mechanical to some extent. It seems that there is a feverish urge to find some perceptual proof in support of the patient's complaints. And if this tangible proof is not found by demonstrating an infection or by some other physico-chemical yardstick the student is apt to feel frustrated and to conclude that the patient is not really ill.

Perhaps a little more time could be given to the teaching that we should regard the patient as a whole man or woman with a home, anxieties and economic problems, a past and a future, and a job to be held or lost.

It is not possible here to speak of the influence emotional upsets may have on bodily function. Suffice to say that the physician will have a number of patients with whom he will inquire into personal situations which may give rise to anxiety, hostility, aggression, guilt, chronic resentment, discontent, and other disturbing emotions and unwholesome attitudes that may act as a cause or an aggravating factor in the production of much ill health, both psychic and somatic.

Your psychologically disturbed patient rarely complains of his anxiety, depression or fears, but he does start off with the presenting physical symptoms such as headache, backache, palpitation and fatigue.

I realize that we are all fearful of labelling an abstruse organic condition as psychological; but provided we have

¹ Read at a meeting of the South Australian Branch of the British Medical Association on February 28, 1952.

listened carefully to the patient, given encouragement here and there, and if need be made a direct inquiry into the emotional troubles of the patient, *plus* a physical examination, we ought not to make many mistakes.

When satisfied that there is a disturbance of the psyche rather than of the soma, do not tell your patient that there is nothing the matter with him and that he should pull himself together.

I should like to point out that we still find that depressives and early schizophrenics are being sent "away for a change", "to the country", "for a holiday", "for a sea voyage", at a time when early treatment can avert tragedies.

Similarly, grossly hysterical patients are subjected to batteries of investigations, the results of all of which are negative, and all for the want of a little psychiatric insight and a sympathetic interest in the patient's human problems, which would perhaps take a fraction of the time and expenditure of all the investigations.

In dealing with your psychologically disturbed patient with psychosomatic symptoms, tell him in unequivocal terms that his symptoms are due to emotional disturbances, and if necessary give illustrations to him.

You know the ordinary physical concomitants of rage, such as increased blood pressure *et cetera*. Now in primitive life the enraged organism attacked, and then the blood pressure returned to normal. Similarly the physical concomitants of fear are many. But if anger, frustration, resentment, fear, anxiety are the constant emotional states of your patient, then those previously mentioned normal physiological reactions now become permanent, and then we have hysterical states, peptic ulcers, loss of weight, undue fatigue, "D.A.H.", effort syndrome, tachycardia, and a lot of other complaints.

Suppose we consider Da Costa's syndrome, which has at various times had such names as "D.A.H.", "soldier's heart", effort syndrome and cardiac neurosis.

I take Da Costa's syndrome not because it is common nowadays, but because it is probably the simplest illustration of a disturbed mental state producing physical signs and symptoms, strongly suggestive of an organic cause.

The symptoms include precordial pain, breathlessness, palpitations, fatigue, dizziness, undue sweating and headache.

This collection spells heart disease to the patient, and although he is undoubtedly ill and the symptoms are real and not imaginary, the heart itself is not organically disordered.

Paul Wood, of the National Heart Hospital, states that in World War I there were 60,000 casualties labelled "effort syndrome", whereas in the recent war there were relatively few, mainly because the medical officers were alerted to the psychogenic origin.

The disorder is very real, but it is usually due to some chronic anxiety in an individual with a poor psychiatric family tree.

The events initiating the overt symptoms are generally those day-to-day stresses and anxieties that the reasonably well-adjusted individual takes in his stride. Of course battle fears and other severe stresses did start off many cases of this syndrome, when otherwise the subjects would have weathered the storms of life.

In civil life some less well-endowed individuals are timid and over-anxious, suffer inferiority feelings and are unduly apprehensive. In such a soil it is not a big step to the psychosomatic manifestations of Da Costa's syndrome and many other psychosomatic disorders.

Treatment.

Having decided that the condition is psychogenic—and for this purpose X-ray and electrocardiographic examinations are justifiable for the purpose of convincing the patient that the heart is not diseased and that he is not in danger of imminent death—you should in unequivocal terms explain that the heart is sound and that the symptoms are due to his unwarranted fears and anxieties working in a vicious circle. This sounds easy, and in

point of fact you will not be entirely successful in many cases, but your aim may well be to ameliorate the condition and keep the patient at his job.

Here, an hour or so spent in investigating the patient's personality, sympathetically discussing his fears and anxieties, may prove very profitable in so far as your time is concerned. Again I would say that the physician must be priest also, and although in many such cases we cannot hope to make the patient a well-adjusted individual, we should aim at giving him a psychological insight into his condition and, if need be, let him lean to some degree upon his doctor.

In those cases in which the emotional disturbances causing the symptoms are not laid bare to the physician, then the aid of deeper mental exploration by the psychiatrist may be advisable.

Many patients suffering from anxiety states are returned to normal emotional health by the particular type of psychotherapy used by the doctor.

This therapy, whether it be the old-fashioned wisdom and deep understanding of human problems by the family doctor, or psychoanalysis of the Freudian, or a Weir Mitchell régime, in each case has its good results, due in no small degree to the doctor-patient relationship.

With my profound ignorance of the wonders of the Freudian doctrine, I still concede its worth in the hands of the enthusiast, even though psychoanalysis has been maligned as "the art of describing the commonplace in terms of the incomprehensible".

I should like to mention also that there are many neurotics whose lives must be "managed" by the doctor to keep them in reasonable health rather than to effect, shall we say, a "cure".

The Early Diagnosis of Schizophrenia.

Schizophrenia covers such a colossal field that it is difficult to give its limitations; indeed, I feel that too many passing deviations from the ideal standard of conduct are thrown into this bin with the label of schizophrenia.

You might recall that formerly this disorder, in its not so early manifestations, was known as primary dementia, emotional dementia, *dementia praecox*, and finally Bleuler coined the word schizophrenia.

The physician considering whether he has a case of early schizophrenia will do well to remember that there are many people who have shown schizoid traits throughout their lives and who may or may not pass on to a definite psychosis.

They are the people who have always been a little "queer"—remote, without any warmth in their interpersonal relations, the bad mixers, those who are looking for "the catch" in ordinary dealings, those apt to ponder upon the imponderable.

Although they may go through life with these characteristics without showing an overt mental disorder, some of this group do develop an acute florid psychosis showing a wealth of fantastic delusions, hallucinations and bizarre conduct.

However, here I want to mention a few of the early manifestations, because if the condition is diagnosed soon enough the prognosis is not so bad.

Now the family doctor is well aware that the passing from youth to maturity is often associated with some oddities of conduct. These oddities are not the result, *per se*, of physiological changes, but rather those stemming from a psychological change from dependence to independence.

These youthful oddities comprise undue sensitiveness, stubbornness, a coquettishness, a lack of warmth in the family circle, sometimes a rebelliousness, unpredictable outbursts of temper, a remoteness, a tendency to seclusiveness and suspicion. All these characteristics fit into the picture of an early schizophrenia, but in most cases they pass away and the youth makes a successful adaptation to his life's environment.

Unfortunately the same characteristics may remain unduly long or become worse, and it is then that the doctor may have to make a diagnosis of schizophrenia.

Perhaps we might pause to consider what is the "normal" or "average" person in everyday life. Well, we find a certain orderliness and harmony in his mental life, and if you divided his personality up in the usual three parts you would find the following.

The man's total personality would be an integrated whole, in which thought is logical and ideas follow one another in related sequence; the feeling is congruous with his thoughts; and what he does is expedient and fitting.

But in schizophrenic disorder we find a disruption of these three parts of the personality—mental life is no longer a harmonious whole. Thinking is not in sequence, there is a dissociation; for example, if at a set moment you recorded the thoughts of two different people on different subjects you would get some idea of schizophrenic thinking. Then feeling is no longer consonant with the thoughts, and finally what the patient does is non-adaptive—it does not seem to fulfil any useful purpose and it is unpredictable.

What are the symptoms which will justify a diagnosis of early schizophrenia?

Firstly, there is a disorder of thought in that the patient's thinking, as shown by his utterances, just does not follow the usual laws of association; his replies in the early stages leave you a little baffled, you wonder just what he means. There is an inconsequence and odd sequence of ideas. He is slightly irrelevant and he just misses the point in conversations.

If the ordinary adolescent individual has some perplexity he can formulate his question in terms that you can understand and give a ready answer. The early schizophrenic is often perplexed by problems removed from those of the normal youth, but he is unable to express clearly what his problems are; he shows a blocking of thought, that is, he half formulates his inquiry and then gives up. The early schizophrenic is finding the outside world menacing, he is failing to perceive reality as it is, he withdraws into himself, he becomes more introspective and remote. At this stage he is troubled, and the loss of affect does not come until later.

This disorder in thinking, in which ideas follow one another in an apparently unconnected sequence, is minimal in early stages. Later you will find a gross dissociation—for example, the patient has so withdrawn from society that he fails to show normal interest in the things he should be interested in and remains at home.

The doctor who is called talks about kings and cabbages in an attempt to make some *rapprochement* with his patient. He asks whether the patient has any complaints and is informed that there are none. He then asks directly of the patient why he has not gone to his usual work. It is then that he may receive an entirely irrelevant reply, such as "Last Thursday week" or "Sitting on a seat".

The second significant observation is the inadequacy and incongruity of affect.

In all other mental disorders, whether they be a depression, a mania, a neurosis, a dementia or even a congenital deficiency, you can feel with the patient; you can, as it were, get on the same wave-length and have some *rapprochement* with your patient.

In schizophrenia you often feel that you are not making contact with your patient; there is no warmth; you are dealing not only with a mechanical robot, but with a robot whose "innards" have slipped a few cogs.

In the early stages it is simply that the normal bonds of emotion between the members of the family have cooled off, so that there is a shallowness of normal affect.

Some early schizophrenics are aware of this defect, are perplexed by it, but cannot understand it, and it needs some skill and patience by the physician to get the patient to talk about it.

In psychiatry generally we must expend much time both in diagnosis and in treatment, and rushing one's hurdles just cannot be done.

This loss of affective response is one of the most valuable signs in the early diagnosis of schizophrenia, especially when it is so contrary to the usual tenor of the patient.

In the incongruity of affect you see entirely inappropriate emotional responses. He may tell you that his mother has recently died, and this with a silly smirk on his face. In the more flamboyant cases the patient states that he has been murdered, drawn and quartered nightly, and all this with as much affect as though he were passing the time of the day.

The third "red light" in diagnosis is the disturbances of volition.

It may be that he just does not do things of his own initiative as he did before. Instead of his getting up at the usual hour his parents have to prod him repeatedly. On having gone to work and done his job in an inefficient mechanical way, he is apt during his leisure hours to contemplate things mystic and fantastic. We find him becoming more dreamy and more aloof. He mingles less with his fellows; he is called lazy. His self-starter has failed, so that he has to be "cranked" by others.

We find, as a useful pointer in diagnosis, the work history of the patient—has he had several different jobs in the last year or so, and what were his reasons for leaving them?

Careful inquiry may lead the patient to tell you that he does not feel in control of his own will, his thoughts or his activities. He may imply or say that he is influenced by other people, that his thoughts are being read, that he is being hypnotized, drugged *et cetera*.

In drawing attention to the early manifestations of schizophrenia, I have not dwelt upon overt delusions, hallucinations or catatonic symptoms and a host of other manifestations of the obvious case. However, I do mention that a wealth of information can be obtained by simply observing your patient during the interview.

How does he approach the doctor? How does he sit? Does he look at you? Does he suddenly shoot a question out of the blue at you? Does he wriggle and brush imaginary *débris* from the desk? Does he grimace or make queer mouth movements?

Does he show the usual embarrassment of the normal youth in asking about matters of a sexual nature? Does he refrain from meeting your gaze and then occasionally cast a furtive glance in your direction?

If you are in doubt about the diagnosis you may decide to give him some counselling and then watch your patient for a while. I know that in a young adolescent it is a serious step to send him to a psychiatrist, for this may simply confirm his fears that he is going mad.

However, do not delay too long, and even if your patient is simply going through the oddities of behaviour associated with mental maturation, the psychiatrist may be able to help.

If your patient shows sufficient deviation from an arbitrary normal to be classed as early schizophrenia, then psychiatry offers the best hope.

Certification.

I would like to devote a little time to the matter of certification, because I know from experience the great need for practitioners to be better informed.

However, I assure you that should a legal action for wrongful certification be taken against many who certify the individual as mentally defective, the certificates themselves would offer little or no defence to the unfortunate practitioner; indeed, they might well be proof of want of care.

I know you all heartily dislike filling in forms, but we must be realists and recognize that next to one's death certificate the doctor's certificate of mental defectiveness may be the worst catastrophe that can befall anyone, especially in the eyes of the man in the street, who is the usual juryman.

Medically, we may know that the certification may be the individual's only salvation, both for himself and for his family.

Now the various statutes relating to mental illnesses, in which the patient requires certification and a restriction

of his civil liberties for some time, have been evolved over the centuries of law making.

Always, however, the personal liberty of the individual stands out preeminently in English law. Upon the doctor rests a very grave responsibility when he certifies a patient as mentally defective. He should then be most careful that what he records as facts observed by him at the time of the examination are without doubt facts indicating mental defectiveness. The certificate is a medico-legal document, preserved for all time, and by law any person having a reasonable claim will be supplied with a copy of the doctor's certificate.

Historically, it is interesting to note that the laws of the Roman Republic about 451 B.C. were cut in tablets of bronze and set up in a public place. These were called the Twelve Tables, and Table V said: "If he is mad, then his clansman must manage his affairs."

In the advancement of care and treatment for the mentally sick person, we have provided, and rightly so, many safeguards not only for the patient, but also for his property.

Nowadays we try to avoid the use of the word insanity. In South Australia the doctor signing the medical certificate for admission to the receiving house or to a mental hospital certifies that so-and-so is apparently defective or mentally defective, respectively. Moreover, the law requires that the doctor shall specify on the certificate the facts observed by him, upon which he based his opinion.

Therefore we should record facts which will satisfy not other doctors, but a jury of laymen, that the individual is mentally defective.

For practical purposes, Dr. Charles Mercier said long ago: "Insanity is a disorder of conduct." And so every practitioner, when contemplating certifying a patient as legally mentally defective, might ask himself: "Does this man's conduct differ, and in what way, from that of the normal man of the same age, rank, religion and education in similar circumstances?"

By conduct is meant everything the man says or does, and what he refrains from doing when doing something is appropriate.

The same conduct may, in different circumstances, be normal or indicative of gross mental disorder.

For instance, if while walking along North Terrace you saw a man, far from clean and down at heel, picking up cigarette butts, which he carefully stowed away in an old tin, the event would attract no more than your passing attention. But if that man was our impeccably dressed, punctilious, precise President, we should indeed have cause for alarm.

If one man punches another on the jaw, the act itself may be the normal consequences of a healthy disagreement. But if a total stranger is similarly attacked because the aggressor is convinced that this stranger is reading his thoughts, or making him experience fantastic feelings, then the act *plus* the reasons for the attack spells mental disorder.

Indications for Certification.

If we have failed to persuade a patient to undertake treatment on a voluntary basis when the voluntary status is advisable, then certification is carried out, when the patient is likely to be a danger to himself or to others. And under this heading we include failure to appreciate that he is in need of treatment, the taking of inadequate food or shelter, suicidal or homicidal behaviour, and incapability, by reason of organic changes, of adequately caring for himself or of being cared for by others.

I wish to quote here from actual certificates and to show that what has been specified by the doctor does not in any way indicate any mental disorder.

1. "He hears voices." This statement by itself is useless, because don't we all hear voices? To be valid it should be: "John Smith states that he hears voices when alone and there is no possibility of voices being heard."

2. "She sees angels floating around." This is not a fact observed. It should be: "She states that she sees angels floating around." That is a fact observed.

3. "He states that his relatives ill-treat him and that they are stealing his property." Add something like "Such statements cannot be reconciled with the known facts", or "Such is not a fact".

Here are some actual and recent examples of medical certificates: "I have not observed him doing anything abnormal nor can I gather any abnormality from conversation, as he can only speak Italian." "He is incoherent." "He has a wild look in his eye." "Appears very dopey. Vacant look about face."

A medical practitioner is vulnerable if he shows carelessness or want of skill in writing a medical certificate of mental defectiveness.

Some thirty years ago £25,000 damages were awarded by a jury against two doctors in England for the wrongful detention of a man. Fortunately this decision was later reversed, on legal technicalities, by the Court of Appeal.

Last year £980 were awarded against a doctor for careless certification. The circumstances were as follows:

On being called to the home to examine the husband, with a view to certification, the doctor found that he had run away. Hearing the reports of the rest of the family, and knowing the patient previously, and knowing that he had been in a mental hospital before, and knowing that on that occasion the superintendent of the mental hospital had stated that though the patient had remitted from his mental disorder, it would be unwise because of possible violence for the patient to live with his wife, the doctor, knowing all this, wrote out the necessary certificate, without actually seeing or examining the patient at the time.

An individual with a paranoid trend is likely to be litigious, and moreover he may find a solicitor having more than a professional motivation in aiding such a patient or ex-patient.

Now, assuming that you have decided that your patient should be treated in a mental hospital or receiving house, there are several ways in which this can be done, but I propose to mention only two.

As a voluntary boarder he may be admitted on his own written request, provided the superintendent thinks that the patient is suitable.

The patient, of course, must be quite capable of understanding what he is doing on a volitional basis.

From experience, we have learned that it is inadvisable to admit certain classes of patients: (a) depressive patients who are suicidal; (b) some psychopaths who are infirm of purpose and have hardly had the examinations completed (these are time-absorbing to the medical staff) before they formally request to be discharged from hospital; (c) chronic alcoholics, who are not suitable as voluntary boarders; (d) those who have come into conflict with the police and think that admission to a mental institution will serve as a good defence for some unlawful act.

Another method, the most usual, is for the patient's relative or friend to submit a written request *plus* the doctor's certificate. (a) In the case of admission to a receiving house one medical certificate is necessary. (b) In the case of admission to a mental hospital two medical certificates are required.

Now this method is satisfactory, provided the patient is not unwilling to go to the mental institution. If your patient is unwilling, and indeed he may be dangerously and violently resistive, then in these circumstances an amendment made in 1945 to the *Mental Defectives Act* should be invoked.

You may recall that the police were extremely reluctant to enter a house for the purpose of dealing with a patient who obviously might be mentally defective. The patient might be threatening, refusing to eat, smashing up his own furniture; but the police took the view that there was no law to say that a man could not burn his own grand piano if he wanted to do so, and although threatening violence, he had in fact committed no offence.

Under the recent amendment, a police officer of or above the rank of inspector may authorize the police to enter the house and take the patient to a mental hospital or receiving house.

Before the inspector (or higher officer) will act, he must be satisfied (i) that the necessary medical certificate or certificates have been completed; (ii) that a request, as required by the *Mental Defectives Act* for the reception of a patient, has been duly completed, together with the statement of particulars; (iii) that a written request has been made to the police for them to act by the person who signed the request under the *Mental Defectives Act*.

The above method is very useful in many cases, and you will find that the police, including the women police, act with discretion and without fuss or show of uniforms.

Some General Remarks.

The teaching of psychiatry is not discussed here, except to mention one aspect.

Most of us know at heart that we are only cheap imitations of what we would like other people to think we are.

However, it is true that in the eyes of the student the "honorary" is, or should be, a great man, and so the student, by the mental mechanism of identification, adopts some of the philosophy of his "honorary". In the past we have heard of "honorary" dealing contemptuously with the genuine neurotic and of referring to the mental hospital as the "nut factory" or the "giggle house". Would it not be better, as far as medical practice is concerned, to engender the concept that psychiatry is the other half of medicine?

We who work in the mental hospitals realize only too well how many failures we have; indeed they may be with us for up to fifty years. But the occasional honorary medical officer who has in the past adopted a facetious adolescent attitude to psychiatry has done untold harm to the student. And after all, are his overall results of treatment any better than ours? On the other hand, I freely admit that in some respects psychiatry has not had both feet on the ground and that it has richly deserved some adverse criticism.

In the practice of psychiatry we can still be aware that the welfare State is full of dangers in the matter of pensions, where a little healthy work is the best medicine.

The doctor should be the last to enable anyone to gain a pension and loss of his soul. I think it better on the whole for the true neurotic to suffer temporarily considerable hardship rather than to throw in the sponge and become more and more dependent and miserable.

Labour is a biological necessity, and the doctor should at all times endeavour to keep his neurotic patients at work.

As regards psychotherapy, a passing mention has been made of this, because every successful practitioner consciously or unconsciously practises it. Indeed, it has been my experience that medical men who stoutly disclaim all knowledge of the art are often very adept in the practice of psychotherapy.

Sound family doctor psychotherapy is something more than either the paternal back-patting "you'll be all right" philosophy or the jargon of the false pseudoerudite doctrines of psychology so popular in the films and Press.

The patient, whoever he may be and whatever he may be suffering from, does much better if he has confidence in his doctor. The experienced surgeon is well aware of this, and he unconsciously assesses the mental state of his patient before he undertakes a serious operation. If the patient's "will to live", as it were, is at a low ebb, the surgeon does postpone the operation, if this is possible, until his patient has recovered a more healthy mental state.

With some patients a healthy detachment on the part of the surgeon may be advisable, while with other patients the surgeon's aloofness may lead to much needless worry where a little simple explanation would dispel the false fears held. Recently in the journals there have been several

references to this matter under the caption of "The Iron Curtain in Hospitals".

In so far as psychotherapy is concerned, I point out that we may regard the personality as possessing a certain, perhaps rigid, structure, with a bundle of "qualities" which has been built over the years by the bilateral operation of the constitutional make-up and the outside world, particularly in childhood and in youth.

What the personality has acquired from the outside world can be modified by the outside world; that is, by psychotherapy certain desirable changes can be brought about. Whether what we call endogenous can be modified by psychotherapy is perhaps debatable. However, you well know that there are appropriate kinds of *milieus* in which the neurotic, the moron, the hypomanic, the schizoid, can lead constructive and productive lives; and conversely certain ways in which the same persons come to grief.

It is pointed out that in these people it is not the constitution itself, but the finding or the not finding of the appropriate environment that determines their destiny, and that is where psychotherapy may be useful.

In this talk I have not referred to any of the special physical methods of treatment used in psychiatry today. I am simply stressing the importance of regarding man as a biological organism, whose path through life is determined not only by physical, chemical, physiological and neurological factors, but also by mental, emotional and social factors.

Frequently the nature of a bodily disorder can be appreciated only when psychological happenings as well as physical disturbances are investigated.

As I see it, the causes of ill health are sometimes mainly physical, sometimes mainly emotional, and it is for the family doctor to assess how much of the one or of the other is operating at the particular time.

In the words of Professor Aubrey Lewis: "Always the rule of rules should be to treat the individual who is sick, not an abstract disease."

No one is better qualified to do this than the family doctor, who does know the family disabilities, both physical and emotional.

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A SHORT-ACTING MUSCLE RELAXANT, SUCCINYL-CHOLINE-CHLORIDE.

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The use of muscle-relaxing agents in anaesthesia is well established and requires no detailed discussion. Until recently no very short-acting drugs have been available and it has been considered by many anaesthetists that such preparations would be useful particularly in laryngeal intubation and in electro-convulsive therapy.

At the Congress of Anaesthetists (1951) recently held in London, two such drugs were reported—namely, "362 I.S." and succinyl-choline-iodide or succinyl-choline-chloride. The arrival in Australia of the last-mentioned has allowed a small series of cases to be studied, and the following notes are the result of experience of about fifty administrations of succinyl-choline-chloride marketed under the name of "Scoline" by Allen and Hanburys, Limited.

"Scoline" is supplied in ampoules, each containing 100 milligrammes of succinyl-choline-chloride dissolved in two millilitres of solvent. The solution is incompatible with thiopentone, a flocculent precipitate being produced on standing. This fact contradicts a statement made at the Congress of Anæsthetists by Dr. Myerhoffer, of Vienna. After intravenous injection, succinyl-choline-chloride produces muscular relaxation by depolarization of the muscle cell membrane. It is destroyed by body esterases and potentiated by neostigmine. It would appear, therefore, that succinyl-choline-chloride behaves, at the end-plate, in a manner resembling that of excess acetyl-choline such as is produced in a normal person by the administration of neostigmine.

Succinyl-choline-chloride, when injected intravenously, first produces muscle stimulation, which appears as action varying from slight twitching of individual muscles to the movement of a whole limb. These effects appear in from thirty to sixty seconds after intravenous injection, last from fifteen to thirty seconds, and are followed by complete relaxation. No side-effects other than a slight rise of blood pressure have been observed, and succinyl-choline-chloride does not cause the release of histamine.

When a clinical trial of "Scoline" was commenced, 25 ampoules, each containing 100 milligrammes of drug, were available. It was decided to study the action in two groups of subjects—namely, those undergoing electro-convulsive therapy and those in whom a rapid return of respiration was desired after laryngeal intubation.

Electro-Convulsive Therapy.

In electro-convulsive therapy it is desirable to modify the convulsion to an extent which will avoid skeletal damage, but will still allow the convulsion to be recognized. Sufficient thiopentone should be administered to ensure that the patient is unconscious when the shock is given, but regains reflexes rapidly after the convulsion. A 5% solution of thiopentone has been employed with a dosage of 0.5 millilitre per stone (six kilograms) of body weight. To this has been added one one-hundredth of a grain (0.6 milligramme) of atropine sulphate. This addition has been found desirable, as without its use salivation appears after the administration of "Scoline". It would appear that the succinyl-choline-chloride does produce some parasympathetic stimulation. The dosage of "Scoline" employed has been 2.5 milligrammes (0.05 millilitre) per stone (six kilograms) of body weight. For the average ten-stone patient, then, the dosages were five millilitres of 5% thiopentone solution and 0.5 millilitre of "Scoline".

Before the technique that has been used in electro-convulsive therapy at the Alfred Hospital is described, it is necessary, in view of a recent article by Webb (1951), to refer to the purpose of oxygen administration with this method of treatment. Webb stated that the virtue of his technique was that, since the muscles of respiration were not paralysed, "apparatus for instituting controlled respiration should always be at hand, but will seldom be needed". Such apparatus must always be used with electro-convulsive therapy. If a patient is rendered apnoeic by any means and the lungs are filled with air, then the patient will become anoxic within about thirty seconds of the onset of the apnoea. However, if the lungs are filled with oxygen before the apnoea occurs, this state can persist for up to seven minutes before anoxia results. Since electro-convulsive therapy will produce a period of apnoea, regardless of whether or not muscular relaxants are used, the patient should be given oxygen to breathe for about two minutes before the shock is administered. The purpose of the oxygen apparatus is not only for the performance of controlled respiration when prolonged apnoea results, but to ensure that the patient's lungs are filled with oxygen before the convulsion.

Technique.

The patient is weighed before the first treatment and the dosage of thiopentone and of "Scoline" is calculated as already stated. Atropine is added to the thiopentone.

With the patient reclining on the couch the needle is inserted into a suitable vein and the thiopentone-atropine mixture is administered. A small quantity of blood is drawn back into the syringe so as to free the needle of thiopentone solution, and the syringe is disconnected. A second syringe, containing the "Scoline", is connected to the needle. As soon as the patient loses consciousness the head-band of the electrical equipment is applied and an apparatus consisting of bag and mask is applied to the face. Oxygen at the rate of ten litres per minute is flowed into the bag.

When the head-band is in position all electrical connexions have been made and the patient has been breathing oxygen for about one minute, the "Scoline" is given. The patient is observed for muscular movements, and as soon as these cease an airway is placed between the teeth, the chin is supported and the shock is given. Apnoea usually lasts for a period of about two minutes and full intercostal respiration has returned within three minutes. If oxygen has been given efficiently, no cyanosis should appear before full respiration is reestablished. With the dosages quoted the convulsions have been well modified, facial twitching and slight movements of the limbs being the maximum disturbance observed.

Laryngeal Intubation.

For laryngeal intubation larger dosages of both thiopentone and "Scoline" are required. In the average adult patient 0.5 grammes of thiopentone followed by 75 milligrammes (1.5 millilitres) of "Scoline" will produce full relaxation of the jaw muscles and loss of laryngeal reflexes.

The anaesthetic agent which is employed following intubation with these agents should be one which produces a considerable depth of anaesthesia rapidly, so that the laryngeal reflexes can be depressed by the anaesthetic agent before the effects of the "Scoline" have passed off. Nitrous-oxide-oxygen-thiopentone, cyclopropane and "Trilene" have all been used with satisfactory results. "Scoline" has proved particularly valuable in dental cases, in which endotracheal anaesthesia is to be followed by the rapid extraction of a number of teeth. The full benefit of rapid induction and intubation can then be followed by the rapid return of the cough reflex and voluntary respiration without the use of neostigmine.

Summary.

1. The action of succinyl-choline-chloride in producing muscular relaxation is discussed.
2. The technique employed at the Alfred Hospital in 35 cases of electro-convulsive therapy is detailed.
3. Laryngeal intubation in 15 cases under thiopentone anaesthesia supplemented with "Scoline" is described.

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OBSERVATIONS ON THE USE OF "SCOLINE" AS A MUSCLE RELAXANT.

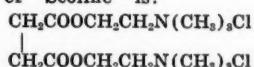
By J. EGERTON WILLIAMS,
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SUCCINYL-CHOLINE-CHLORIDE, or "Scoline", is the latest muscle relaxant to come to the assistance of the anæsthetist. It is remarkable for its ultra-rapid action, whereby a relatively small dose presents a profound relaxation lasting for two to three minutes only, after which time the respiratory muscles contract again and breathing commences to become adequate in a further minute.

The action of "Scoline" is a neuro-muscular block by depolarization, there being a stimulation before paralysis

that accounts for the minor seizure which occurs within five seconds of injection. Details of this type of action were explained by Feldberg in the *British Medical Journal* in May, 1951.

The formula of "Scoline" is:



First reports on the muscle relaxant qualities of "Scoline" came from Bovet (1949) and later from others, the first clinical report of its use on an anaesthetized patient being by Brücke in 1951. "Scoline" is made up in a sterile colourless solution of 100 milligrammes in 2.0 millilitres; it is destroyed by alkali, hydrolysis occurring in a short time (three to four minutes), indicating that "Sodium Pentothal" and "Scoline" should not be used together in the same syringe unless the requirements mentioned below are fulfilled. When "Pentothal" and "Scoline" are injected from different syringes but through the same needle, some degree of mixing and contamination is bound to occur, so that if possible different veins should be used.

"Sodium Pentothal" and "Scoline" can be mixed provided (a) that the injection is carried out in under three minutes, that is, before any hydrolysis could occur; and (b) that there is a marked excess of "Pentothal".

Fulfilling these requirements, we have evolved a technique which has now been used in 247 cases without any complicating factors or side effects. The arm must be prepared beforehand and the vein chosen. Should any difficulty be expected, such as hidden veins, the mixture of the substances should be avoided. In cases for electric convulsive therapy where several patients are to be treated immediately after one another, we have syringes with the required dose of "Pentothal" already drawn up, placed in position on the anaesthetic table, and the "Scoline" is drawn up into the syringe literally at the last second. The "Scoline" shows itself in the syringe as a white cloud which immediately disappears owing to the excess of "Pentothal", and to assist this we have drawn up two millilitres of air and given a sharp shake before the air is expelled. Provided there is now no precipitate, the injection may be made at average speed, the needle withdrawn and inflation of the lungs with oxygen commenced.

Within five seconds of injection with "Scoline", whether mixed or not, there is a marked diffuse contraction of all muscles, signifying the stimulation caused by the "Scoline" reaching the myoneural junction. These contractions last five to ten seconds and then cease altogether, indicating complete paralysis. (It has been noted that when "Scoline" is given during an operation in which anaesthesia has been in progress for some time, and other relaxants have been in use, the diffuse contractions are almost unnoticeable, if visible at all.) The intended procedure may now be undertaken following oxygenation.

A series of experiments were carried out by mixing "Scoline" and "Pentothal" drop by drop, then millilitre by millilitre, in order to prove the importance of excess of "Pentothal" in delaying the onset of hydrolysis. When the volumes were equal, hydrolysis was immediate and complete, but as the ratio of "Pentothal" to "Scoline" increased, the hydrolysis took longer to appear; so that, when the approximate 8 to 1 ratio of the average injection for this type of anaesthesia was reached, fully three to four minutes elapsed before hydrolysis occurred.

The Uses of "Scoline".

The uses of "Scoline" may be classified as: (a) for intubation, (b) for electro-convulsive therapy, (c) for manipulations, (d) during operations in which rapid but short relaxation is required.

Intubation.

The use of a relaxant to assist in intubation was first described by Bourne (1947). "Scoline" when used for this purpose presents a more profound relaxation than previous substances of this nature. The view of the cords and

larynx is maximal and an endotracheal tube may be passed quickly and certainly without trauma of any kind. Spraying of the vocal cords, if necessary, is greatly facilitated by the complete relaxation. When further relaxation is required later in the operation, a more lasting relaxant should be used, although "Scoline" can be given in small repeated doses with satisfactory result but without the "smooth" quality of the anaesthesia. When spontaneous respiration after intubation is required this method is ideal.

Of the intubations carried out by us, 64 were dental clearances at Repatriation General Hospital, Heidelberg, intubation being essential, but adequate breathing being required as soon as possible. Provided no trouble was encountered, this operation took only four to five minutes, and since most patients were well-built young men, the required dose of other relaxants to allow intubation would present the anaesthetist with a curarized patient at the end of the operation. When trouble was encountered, such as broken roots, impaction, or haemorrhage, small repeated doses of "Scoline" were given with excellent results, the anaesthetic being continued with nitrous oxide and oxygen if necessary. With some cases there seemed to be more haemorrhage after the use of "Scoline" than with other methods of anaesthesia; and it was during these operations that the rapidity with which the patient can "come out" of both anaesthetic and relaxant was noted. It should be emphasized here that adequate anaesthesia must always be given even if the relaxation suddenly becomes insufficient, and never should "Scoline" be injected before the "Pentothal", since its action is so very rapid.

Electro-Convulsive Therapy.

When "Scoline" is used the type of convulsion seen is much milder than that with other relaxants owing to the profound paralysis. The first initial jerk as the current flows is obvious, though less marked; while the actual convulsion itself is in many cases difficult to see at all. This fact is one of the disadvantages of "Scoline" in this work, although the advantage of rendering the patient safe from trauma or fracture is all-important. The tonic spasm can generally be seen as an uplifting of the eyebrows or a turning in of the toes, whereas the clonic spasm shows itself as a twitching of the body muscles, best seen as a fibrillary movement of the facial muscles.

Manipulations.

For many manipulations a relaxant is unnecessary, although manipulation of the spine in a well-built individual can seldom be done without some assistance. Some surgeons have said that the relaxation is so great that they feel they may do damage, and would rather use their own strength as a guide than have the patient too flaccid. In manipulations of the feet, spasm of the calf muscles often occurs, and this complication may ruin the effect of the manipulation; it can be controlled by "Scoline".

Other Uses in Surgery.

Under the heading of "other uses" are instances during operative surgery where relaxation is needed quickly, though for a short time only.

Closure of the Peritoneum.—During closure of the peritoneum, particularly where there has been a long operation and quite large doses of other relaxants have been given, the anaesthetist being anxious not to give more respiratory depressants at this stage of the operation, "Scoline" may be used. A similar dose to that used in intubation will give all the relaxation required and will have worn off in four to five minutes without any accompanying respiratory depression. A case may be quoted.

Hysterectomy was performed on a woman of 11 stone, the operation had been in progress two hours and she had received a total of 200 milligrammes of "Flaxedil" during the course of that time; to close the peritoneum 0.5 millilitre (25 milligrammes) had little or no effect and it was not until a further 75 milligrammes had been given that the required relaxation appeared. We had expected that relaxants would

have been cumulative, and that a very small dose would have been sufficient, but in most cases quite large doses have been required.

Reducing Dislocations.—Examples of dislocations reduced have been those of the jaw, the shoulder and the knee.

Fractured Femur.—When in a fracture of the femur there is a definite displacement in a muscular patient, the surgeon may find it almost impossible to approximate the bone ends, and a few minutes of relaxation will allow the clamps to be put in position.

When Muscles Are Too Fully Formed.—Fully formed muscles may be a hindrance to the operation. A case in point was that of a man of forty-four years of age, weighing fourteen and a half stone, undergoing a laminectomy. His back muscles were sufficient to prevent the retractor being fully inserted.

Sewing up the Patella.—After fracture and repair of the patella the vasti muscles may be too strong to allow adequate approximation.

Dose.

The dose of "Scoline" must inevitably be related to the dose of "Pentothal" and also to the age, weight and physical condition of the patient. We have found that the doses suggested by the makers are too large and that they can be reduced by a third. As a rough guide we have used: (a) for a large adult, 1.5 millilitres; (b) for an average adult, 1.0 millilitre; (c) for a small adult, 0.5 millilitre.

We have never used "Scoline" for children, though it is said to be satisfactory. When the patient is old or too ill we have adjusted our dose according to clinical finding. A case in point for electro-convulsive therapy was that of a man with tuberculosis of the spine, seventy-one years of age, and weighing seven and a half stone. We found less than half a millilitre gave adequate relaxation. The dose of "Pentothal" required in all cases was sufficient to put the patient just to sleep, the rapidity with which "Scoline" wears off making certain that the patient should not wake first.

Antidote.

Owing to the rapid action of "Scoline" no antidote is required, though it should be reported that neostigmine is contraindicated.

Side-Effects.

We have found no side-effects that could be attributed to "Scoline". The blood pressure falls a little after the injection and appears to return to an even higher level than before. However, since "Scoline" is generally used with "Pentothal", which is in itself a notorious hypotensive, it is difficult to tell whether the "Scoline" is the cause. The pulse rate rises a little, but soon settles. There is evidence that "Scoline" may be used with any anaesthetic without any complicating features. There appears to be more capillary oozing associated with the use of "Scoline". When it was first used for dental clearances the sisters in the wards reported some post-operative bleeding, but this has not shown itself often enough to arouse comment.

We have been careful to ask patients of their impressions during induction of this type of anaesthesia, particularly those who have had repeated treatments such as electro-convulsive therapy, but we have been unable to find out anything that could be recorded. In 247 cases of electro-convulsive therapy inductions were carried out with "Scoline" and "Pentothal" mixed in the same syringe, in different syringes, but given through the same needle, and injected through different veins, and no side-effect of any kind has been encountered.

Conclusions.

A report based on the use of "Scoline" as a muscle relaxant in 412 cases is submitted. Of the administrations, 247 were for electro-convulsive therapy, 64 for dental clearances, 46 for other intubations, 32 for manipulations, and

23 for various surgical operations in which added relaxation was of assistance to the surgeon. "Scoline" gives a more profound relaxation than any other relaxant. No complications or side-effects of any kind were encountered. In over 200 of these cases the "Pentothal" and "Scoline" were mixed in the same syringe. The dose suggested by the manufacturers appears to be too high and might be reduced by one-third. The convulsion with electric therapy is less visible than with other relaxants, and is often difficult to determine at all. Increased capillary oozing has appeared to follow the use of "Scoline" in some operative procedures. "Scoline" is the ideal relaxant for a quick, short-lasting relaxation. Adequate oxygenation of the patient before, during and after the paralysis is essential.

Some anaesthetists have mentioned that "Scoline" takes longer to wear off than has been suggested, but no cases of this nature have been encountered. "Scoline" is manufactured by Allen and Hanburys, Limited, who have done everything possible to assist with this series.

Acknowledgements.

My thanks are due to the Repatriation Commission for access to patients at the Repatriation General Hospital, Heidelberg, and to Mr. A. Parker, of the Facio-Maxillary Unit, for his help and encouragement with the dental cases; also to Dr. Norman R. James, of the Royal Melbourne Hospital, for his helpful criticism, and to Dr. B. Mulvany, Dr. G. Reynolds, and Dr. C. Poustie for their assistance.

Addendum.

Since this article was submitted, a case has been encountered illustrating the small dosage of "Scoline" required for a very heavy man.

The patient weighed twenty-six and a half stone and underwent 19 treatments of electro-convulsive therapy. The required dose of "Scoline" was carefully noted, and it was found that the optimum was only one millilitre (that is, 50 milligrammes). This gave excellent relaxation provided treatment was given within one minute of the appearance of the minor seizure; otherwise the effects of the "Scoline" would have begun to diminish.

Reports of Cases.

ACUTE OESOPHAGITIS.¹

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ACUTE OESOPHAGITIS may result from such causes as the spread of infection from the pharynx, the swallowing of corrosive fluids, radiotherapy *et cetera*, but the most frequent cause is the regurgitation of gastric contents.

Even debilitating illnesses will result in incompetence of the oesophageal opening in the diaphragm, or such incompetence may occur as a congenital defect. Increased intraabdominal pressure due to pregnancy or other causes may result in regurgitation up the oesophagus and thus in oesophagitis. Such increased pressure may initiate a hiatus hernia, which further impairs the competence of the oesophageal opening in the diaphragm. In other cases hiatus hernia or a short oesophagus is associated with regurgitation in the absence of increased intraabdominal pressure. So also, an indwelling gastric tube may be associated with regurgitation into the oesophagus. Oesophagitis due to the regurgitation of acid may be followed by peptic ulceration, and there may then be further inflammation around the ulcer.

Normally the oesophagus opens obliquely into the cardia, but this obliquity is lost in the presence of a sliding

¹ This case was presented at a meeting of the Section of Surgery of the New South Wales Branch of the British Medical Association on October 9, 1951.

hernia. On the other hand, many patients with a para-oesophageal hiatus hernia are free of dysphagia, and its appearance in such patients suggests the onset of oesophagitis or oesophageal peptic ulceration. As Allison (1949) has pointed out, the oblique entry of the oesophagus into the stomach is usually maintained in these patients, and regurgitation does not always occur. Occasionally an oesophageal peptic ulcer is found in patients in whom there is no evidence of regurgitation from the stomach. In these cases it would seem that the ulcers have arisen in heterotopic islets of gastric mucosa.

Regurgitation also occurs after oesophago-gastrostomy for the treatment of carcinoma of the oesophagus or of cardiospasm. In fact, Wooler (1949) states that the peptic ulceration produced by regurgitation after such an operation is a worse lesion than the original cardiospasm.

Scott (1945) described the case of a patient suffering from "idiopathic dilatation of the oesophagus", who was treated by gastrostomy, but in whom a gastric ulcer was subsequently found. Partial gastrectomy was followed by cure of the cardiospasm. He concluded that the condition was a reflex spasm of the lower end of the oesophagus due to the gastric ulceration. No obstruction of the cardiac orifice was found at operation. In this case it is more likely that the condition was due to oesophagitis caused by regurgitation.

The chief symptoms of oesophagitis and of peptic ulceration of the oesophagus are dysphagia and pain or discomfort beneath the lower end of the sternum or in the epigastrium. The pain may be aggravated by lying down and is often relieved by alkalis. In a series of 507 cases Allison (1949) found that oesophagitis and peptic ulceration of the oesophagus were second to carcinoma as the cause of dysphagia in patients aged over fifty years.

Although most patients with oesophageal peptic ulceration pass occult blood in the faeces, haematemesis is not common.

The classical syndrome of oesophagitis may be stated to be pain and dysphagia, whereas that of peptic ulceration of the oesophagus is similar with the addition of haemorrhage, overt or occult.

In the absence of an ulcer "niche", irregular spasm of the lower part of the oesophagus together with hyperchlorhydria will suggest oesophagitis, and this may often be confirmed by oesophagoscopy.

If the correct diagnosis of oesophagitis is overlooked, the patient is usually regarded and treated as having a gastric or duodenal ulcer. Indeed, in some cases such a second ulcer may even be present. In a series of 31 patients with a short oesophagus, Rennie, Land and Park (1949) found that seven had a history of peptic ulceration of the stomach or duodenum. As Wangensteen and Leven (1949) point out, "in all patients with obstructive duodenal ulcers and vomiting, oesophagitis must be common".

Clinical Record.

Mr. T.L.S., aged forty-nine years, having been referred to me by Dr. W. Corlis, was admitted to Sydney Hospital on August 4, 1951, complaining of "heartburn" of two weeks' duration, and of hiccup and dysphagia for both solids and fluids of five days' duration. Relief of the heartburn had been obtained by alkalis, and the hiccup and the dysphagia had gradually subsided. Apart from these symptoms he had been well. He had lost no weight. There was no history of dysphagia or dyspepsia in the past, and he had been working as an electric train driver. His general condition was good, and no abnormality was found on physical examination. His haemoglobin level was 16.4 grammes per centum. The findings on a fractional test meal examination were within normal limits. X-ray examination with a barium bolus revealed "an irregular outline of the lower half of the oesophagus which became grossly distorted and irregular towards its distal portion. The passage of the bolus through the oesophagus was delayed. The oesophagus ended short of the diaphragm and the lower inch was lined by gastric mucosa." Although there was a slight resemblance to "corkscrew oesophagus", it was concluded that "the appearance was consistent

with an extensive medullary type of carcinoma involving the lower half of the oesophagus". No other abnormality was found in the stomach or duodenum. Oesophagoscopy examination showed the mucosa of the lower part of the oesophagus to be pale pink and oedematous, but no ulceration or malignant change was seen. The lowest inch of the oesophagus did not appear to be lined by gastric mucosa. There was no stenosis or dilatation of the oesophagus and there was no redundancy of the mucosa.

Despite the absence of gross abnormality on oesophagoscopy examination it was thought that exploratory thoracotomy was indicated lest a growth be overlooked at a time when the patient would have withstood an oesophagectomy.

At operation on August 7, 1951, in association with Dr. R. J. Malcolm, a synchronous combined thoraco-abdominal approach was made. Adhesions between the base of the left lung and the diaphragm were freed, and the stomach and the lower two-thirds of the oesophagus were displayed; no apparent abnormality in these two viscera was revealed. The oesophageal orifice in the diaphragm was not dilated, and even though the barium bolus examination had revealed gastric mucosa for an inch above the diaphragm, the muscular wall of the oesophagus in this region was identical with that higher up.

Convalescence after the operation was uneventful.

On August 22 X-ray examination with a barium bolus, carried out by Dr. H. G. Marsh, revealed a free passage of the bolus into the stomach. The oesophagus was reported "as being short and there were irregular writhing contractions in its distal third. There was also the appearance of a small hiatus hernia of the stomach."

Since then the patient has been taking an "ulcer diet" with antacids, and has been sleeping propped up. There has therefore been a considerable decrease in his symptoms, and these measures will be persisted in while the improvement continues. If the symptoms increase in severity gastrectomy will be required.

Discussion.

Hurst (1941) has emphasized the value of raising the head of the bed of patients suffering from a congenitally short oesophagus to limit regurgitation through the incompetent cardiac sphincter, and this measure, together with routine antacid treatment, is advisable for all patients with regurgitation into the oesophagus, whatever the cause. Some patients will have already noticed that the pain is aggravated by lying down, or that relief is obtainable with antacids or milk or other foods.

For the patient with oesophagitis who has developed oesophageal stenosis, Allison (1951) recommends reduction of any sliding hernia, the division of the extended phrenoesophageal ligament, and the repair posteriorly of the distended oesophageal hiatus. As this author points out, the retention of the stomach in the abdomen is not sufficient alone to cure the oesophagitis unless the normal sphincteric action of the oesophageal opening in the diaphragm is restored. On the other hand, in the presence of oesophageal stenosis he states that there is little or no alternative to oesophago-jejunostomy, the latter being combined with gastric resection or exclusion, depending on the presence or absence of gastric or duodenal ulceration.

Treatment of a chronic oesophageal ulcer is advisable, for it may lead to ascending fibrosis, to oesophageal stenosis or to haemorrhage, or malignant change may take place.

Castleton and Dolowitz (1948) reported the case of a patient with a bleeding oesophageal ulcer who underwent a gastric resection before the site of the lesion was determined. They concluded that had an oesophagoscopy examination been carried out before the operation, the bleeding could probably have been controlled, and that radical gastric surgery might have been avoided. While oesophagoscopy was required for the local control of the bleeding, the gastric resection and the post-operative ulcer régime were followed by the healing of the ulcer. Other authors, such as Wangensteen and Leven (1949), suggest that even when the correct diagnosis of peptic ulceration of the

oesophagus without stenosis is made, the best treatment is an extensive gastric resection. They report such a case in which a gastric resection was performed on a patient with oesophagitis in order to reduce gastric secretion and acidity, and in order to accelerate gastric emptying. The dramatic improvement in their patient after this operation was without doubt due to the effect of both of these factors, and they point out that either factor alone is inadequate.

Such a gastric resection for these patients has much to recommend it, and the problem of the frequently associated gastric or duodenal ulceration is overcome. However, it is suitable for use by itself only in the absence of oesophageal stenosis.

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A CASE OF CEREBRAL HÆMORRHAGE WITH UNUSUAL FEATURES.

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MRS. M.W., aged sixty-two years, was admitted to the casualty department of the Hornsby and District Hospital on August 4, 1951, at 8 p.m. She gave a history of having swallowed a chop bone, which became stuck in her throat, while eating her evening meal. This provoked an immediate attack of violent coughing, which failed to dislodge the offending piece of bone. Various "home remedies", such as vigorous thumping on the back and putting a finger down the throat, were then tried, but met with no more success than the paroxysm of coughing. The patient was then brought to the casualty department.

On examination the patient was seen to be a woman who looked ten years older than her stated age. Examination of the mouth, fauces, tonsillar fossæ and oro-pharynx failed to reveal any evidence of foreign body. When asked where exactly she felt the obstruction to be, the patient pointed to the region of the cricoid cartilage. No attempt was made at his stage to examine the larynx or hypopharynx.

There were no signs of respiratory distress, but the patient had obvious difficulty in phonating and was hoarse. It was thought that the foreign body was in the larynx, obstructing the vocal cords, but leaving sufficient room for an efficient airway.

The honorary ear, nose and throat surgeon was notified, and arrangements were made to carry out direct laryngoscopy and bronchoscopy under local anaesthesia at 9.30 p.m. The local anaesthetic agent used was a mixture of equal quantities of cocaine hydrochloride 10% solution and adrenaline hydrochloride 1:1000 solution; it is not possible to give the exact total quantity used. The patient's larynx and pharynx were sprayed with the local anaesthetic agent some twenty minutes prior to operation, and again immediately before the examination was carried out.

The patient was given morphine sulphate, one-quarter grain, and atropine sulphate, one one-hundredth grain, by

hypodermic injection at 9 p.m. At 9.30 p.m. the patient was taken to the theatre, and the pharynx, larynx and trachea down to the carina were examined endoscopically. No foreign body was seen, nor was there any evidence of an impacted foreign body having been recently present. The oesophagus was then examined under direct vision, but still with negative results. The only positive finding so far had been a rather moist larynx and trachea despite adequate premedication. This cleared for a time on aspiration.

At this stage it was noticed that the patient's colour was deteriorating, and endoscopy was temporarily suspended. Examination of the patient revealed dilated pupils, a moderate degree of cyanosis, weak pulse and reappearance of a moist airway.

It was obvious that the patient was fast becoming moribund. Laryngoscopy was again performed and intubation of the larynx carried out. Eight to ten ounces of pink, frothy sputum were aspirated from the lungs. Intratracheal oxygen administration was started and the patient was given one millilitre of "Coramine" by intramuscular injection, but she died five to ten minutes later, at 10.20 p.m.

At post-mortem examination a very extensive intraventricular cerebral haemorrhage was found. Unfortunately it was not possible to locate the site of origin of the haemorrhage.

The patient had not been treated previously at this hospital, but it was found after death that she had been an alcoholic for five years until four years before her death. She had been treated by her own doctor some six weeks before her death for some minor disturbance of functional origin. At that time there was no evidence of hypertension.

Discussion.

The interesting point is, of course, whether the woman really swallowed a chop bone, or whether it was just an extraordinary coincidence that, exactly at the time of swallowing a bolus of meat, an intracranial blood vessel ruptured. Presumably the hemorrhage started in the region of the floor of the fourth ventricle, first affecting the nuclei of the ninth and tenth cranial nerves, and thus creating an apparent sensation of laryngeal obstruction.

Unfortunately, a full clinical examination was not carried out when the patient was first examined, so it is not possible to say whether any evidence of a central nervous system lesion existed at that time.

To the casual reader it may appear that a "howler" has been committed, but it is impossible to recapture, in black and white, the atmosphere existing at the time of the incident. It is only in retrospect that one can appreciate the full significance of the case as it presented.

It would be appreciated if any reader of this article has a more learned and/or satisfactory explanation to offer.

Acknowledgements.

I wish to thank Mr. F. A. Naveau, secretary and chief executive officer, Hornsby and District Hospital, and Dr. A. L. Clowes, honorary consultant ear, nose and throat surgeon of the hospital, for permission to publish a report of this case, and Dr. F. A. E. Lawes, honorary consultant physician of the hospital, for his help in compiling this report.

Reviews.

PATHOLOGICAL HISTOLOGY.

"PATHOLOGICAL HISTOLOGY", by Robertson F. Ogilvie, was first published in 1940. In 1951 the fourth edition¹ appeared, ample evidence of the popularity and usefulness of this excellent aid to the study of the microscopic changes to be

¹ "Pathological Histology", by Robertson F. Ogilvie, M.D., Drennan, M.D., F.R.C.P.Ed., F.R.S.E.; Fourth Edition; 1951. Edinburgh: E. and S. Livingstone, Limited. 8½" x 6", pp. 518, with 295 illustrations in colour. Price: 40s.

found in the organs and tissues of the human body in the various diseases to which it is prone. In the latest edition the mode of presentation remains the same as in earlier editions. An outstanding feature of the book is the inclusion of 295 figures in colour. The text consists of a description of the microscopic changes illustrated in the figures, and these histological descriptions are supplemented by accounts of the gross appearances of the lesions. The paper seems to be better in this edition than in the last, and the illustrations appear to be more distinct. There are 37 more illustrations in this edition and some of the old ones have been replaced. The number of pages has been increased from 447 to 494. It should always be kept in mind that the best way to study pathological histology is by microscopic examination of histological preparations by the student, whether undergraduate or graduate; Ogg's book, however, will continue to prove a very valuable companion to such study. It is a credit to all concerned with its publication.

EAR, NOSE AND THROAT TREATMENT.

In the "Modern Medication of the Ear, Nose and Throat", Noah D. Fabricant attempts to meet the day-by-day requirements of those practitioners interested in the practical application of medication of the ear, nose and throat.¹ He aims at presenting both recent advances and time-tried experiences with drugs for the specialist as well as for the general practitioner and points out that in an era of uncontrolled usage of antibiotics there is greater need than ever for intelligent therapeutics.

The subject matter of the book is presented in an easily read manner. As a background a good deal of emphasis is laid on physiology, anatomy, histology and pathology; at the same time the author draws on clinical experience. A great deal has been compressed into a small volume, but nevertheless it must be admitted that it embraces only the commoner conditions met with in the practice of the oto-hypo-laryngologist.

Two particularly good chapters are those on "Nasal Physiology and Bacteriology" and "Physiologic and Histologic Effect of Drugs" within the nose, especially the latter, in which he clearly defines the limits to be applied in rational medication.

So far as it goes, the book is very sound in the principles of treatment as well as in other respects mentioned above. However, its usefulness is somewhat limited firstly by the fact that it embraces the therapy of only the commoner disorders, and secondly by the fact that numbers of the drugs mentioned are unobtainable in this country. Twenty figures illustrate the text and there is a full bibliography at the conclusion of each chapter.

IRISH MEDICAL HISTORY.

It seems that most countries in the past have experienced difficulty in finding a person with the necessary enthusiasm, ability and artistic insight to undertake the stupendous task of compiling, and then suitably presenting, a complete record of its medical history. In Ireland, Dr. John Fleetwood has gone to the trouble of collecting a mass of material with this object in view and has set it out in a book, "History of Medicine in Ireland", which should be of value to students of the subject, though it may fail to grip the interest and close attention of readers beyond the Emerald Isle.²

In the earlier chapters reference is made to some archaeological evidence of pathological conditions found in human remains from the native soil; inferences are drawn from legendary accounts of medical activities in the Gaelic literature of pagan times; and an irregular assortment of historical information to do with medical care covers a period beginning with the advent of Saint Patrick in A.D. 432 through the influences exerted on Irish medical practice by such mediæval authors as John of Gaddesden, until we come almost imperceptibly to an age of enlightenment. The book is concerned mostly with an account of the medical

¹ "Modern Medication of the Ear, Nose and Throat", by Noah D. Fabricant, M.D., M.S., with a foreword by Austin Smith, M.D.; 1951. New York: Grune and Stratton. 9" x 6", pp. 262, with 20 illustrations. Price: \$5.75.

² "History of Medicine in Ireland", by John Fleetwood, M.B., D.P.H.; 1951. Dublin: Browne and Nolan, Limited. 8½" x 6", pp. 432, with 17 illustrations. Price: 21s.

practitioners and institutions which have helped to mould the future developments of medical practice in Ireland; but hardly enough emphasis is given to the social, political, scientific and philosophical backgrounds to make it really interesting.

In his foreword, Dr. William Doolin affirms that a definitive history of Irish medicine has yet to be written; and whatever truth there may be in the statement, Dr. Fleetwood's initial venture into the larger sphere will undoubtedly serve a useful purpose when the time arrives for it to be written.

RECENT ADVANCES IN CLINICAL PATHOLOGY.

THE second edition of "Recent Advances in Clinical Pathology" is a work which worthily supplements the excellent first edition.¹ The format is approximately the same, that is, the work is subdivided into four sections, each under the direction of an editor. The section editors, with the exception of Dr. B. L. Della Vida, whose hematology section has been taken over by Dr. R. G. Macfarlane, are the same as in the first edition. Several chapters of the first edition have been completely rewritten and expanded, in most cases by the original contributors.

The contributions on cytology have now been placed in the section of histology instead of hematology, an arrangement which seems more logical. In the bacteriology section a chapter has been included on fungous infections. This is very welcome in view of the increasing recognition of the importance of fungi as pathogenic organisms.

Chemical assay of hormones in the urine (biochemistry section) describes procedures for measurement of excretion of 17-ketosteroids, pregnandiol, and corticosteroids, and deals briefly with their clinical significance. The author states that their use at the present stage of development is rather to supplement than to replace biological tests hitherto in use. One is, however, slightly disappointed that the Gallini-Mainini pregnancy test, with its many advantages, has been completely overlooked.

The chapter upon the detection of radiation over-exposure (haematology) is of such obvious value as to require no further comment. The contribution on histochemistry in diagnostic histology will be of more interest to the research worker than to the routine histopathologist, in spite of the undoubted fact that the techniques often reveal unsuspected detail of diagnostic value.

A special chapter on laboratory design and equipment is a most useful adjunct, and will be of great interest to designers of laboratories, whether hospital size or modest one-man establishments.

To sum up, one can do no better than to quote from the preface: "Only one quarter of the subjects dealt with in the first edition have found place in the second, and the chapters dealing with these have been largely rewritten. This does not mean that the subjects of the remaining three quarters of the chapters in the first edition have ceased to be of interest to Clinical Pathologists, nor that these chapters are no longer of value." This useful book deserves a place in the library of any medical practitioner, alongside the equally worthy first edition.

HANDBOOK OF DISEASES OF THE BLOOD.

IN an epilogue to his new book,² Dr. A. Piney writes: "It is customary to lament the increase of specialism in medicine, and this process is at least as striking in haematology as in any other branch. It is for this reason that books on haematology have become larger and larger and more and more incomprehensible to the practitioner of medicine." Accordingly, he has made what is, he believes, the first attempt to produce a practitioner's handbook of

¹ "Recent Advances in Clinical Pathology", by various authors. General editor: S. C. Dyke, M.D. (Oxon.), F.R.C.P. (London). Section editors: Bacteriology, R. Cruickshank, M.D. (Aberdeen), F.R.C.P. (London); Biochemistry, E. N. Allott, M.D., B.Sc. (Oxon.), F.R.C.P. (London); Haematology, R. G. Macfarlane, M.D. (London), M.R.C.P. (London); Second Edition; 1951. London: J. and A. Churchill, Limited. 8" x 5½", pp. 588, with 37 plates and 36 text figures. Price: 40s.

² "Handbook of Diseases of the Blood", by A. Piney, M.D., M.R.C.P.; 1951. London: Harvey and Blythe, Limited. 9" x 6", pp. 224, with 11 illustrations. Price: 21s.

blood diseases. Piney is well equipped for this task, as he is the author of several books on haematological subjects and has practised for many years as a "physician-haematologist". His previous books are commendably simple and unpretentious; the present one is no exception to this rule. It is perhaps an over-simplified account of the subject; but it is eminently readable and may be of more use to the busy general practitioner than larger and more complete works. In his preface the author laments the fact that the diseases of the blood and blood-forming organs have tended more and more to become the province of the clinical pathologist. This state of affairs, he thinks, has arisen as a result of the feeling that bedside observation of patients with blood dyscrasias gives less information and that of less importance than does laboratory investigation. It is, of course, obvious that the laboratory is essential for the diagnosis and control of treatment of these maladies; the point Piney wishes to make is that the patient must be considered as a whole, and that the physician must be able to perform the fundamental laboratory tests and to assess the results thereof. The first step is a careful clinical examination; this is the background against which laboratory findings must be viewed. All will agree with Piney that it is dangerous to divorce laboratory tests from the clinical picture; but in the endeavour to write a clinical handbook, he has perhaps departed too far from scientific precision. Except for the last chapter there are no references to the literature; the opinions expressed are presumably all those of the author. The illustrations are interesting. They include pictures of normal red cells, spherocytes and target corpuscles by the method of "embrage", radiograms of the bones in osteoporosis, myelomatosis and Gaucher's disease, and colour plates of the *fundus oculi* in achrestic anaemia, aplastic anaemia, thrombocytopenic purpura, leucæmia, Hodgkin's disease and poly-cythaemia.

LIVER DISEASE.

FOLLOWING the development of biochemical tests and the introduction of needle biopsy of the liver during the past decade considerable advances in our knowledge of the function and structure of the liver in health and disease have taken place. Many of these advances are very well described and illustrated in "Liver Disease", a Ciba Foundation Symposium, edited by Sheila Sherlock, of the Post-graduate Medical School, London, and G. E. W. Wolstenholme, of the Ciba Foundation, London.¹ The Foundation is to be congratulated for collecting in London the leading "hepatologists" of the world for free discussion. In "Liver Disease" are recorded the highlights of their debates. Although the book will be of particular value to the morbid anatomist, the physiologist and the biochemist, the physician will find much to interest him: for the chapters include "Recent Development in Ficollation Tests" by MacLagan (England), "Amino-acid Metabolism in Liver Disease" by Dent (England), "Changes in the Constituents of the Liver Cell in Early Choline and Protein Deficiencies" by Kosterlitz (Scotland), "Experimental Hepatic Cirrhosis" by Glynn (England), "Effects of Antibiotics and Vitamin B₁₂ in Cirrhosis and Necrosis of the Liver" by György (United States of America), "Needle Biopsy Studies of the Liver in Cirrhosis and other Conditions" by Schiff (United States of America), and "Portal Hypertension in Cirrhosis of the Liver Treated Surgically by Portacaval Shunt" by Gammeltoft (Copenhagen).

A fascinating chapter comes from the pen of Børneboe, of Copenhagen, on the effect of the suprarenal cortical hormone, cortisone, on the production of antibody with special reference to the production of γ globulin in chronic infectious hepatitis. He has shown that the hormone has an inhibiting effect on the formation of antibody globulin. The hormonal control of resistance to infection may have an important bearing on the course of infectious diseases.

The epidemiology of infectious hepatitis is discussed by Joseph Stokes, of Philadelphia, one of the greatest epidemiologists of modern times. As the virus of infectious hepatitis will not definitely grow in experimental animals, Stokes has resorted to the use of human volunteers for his experiments. He discusses the prevalence and the symptomatology of infection by this virus and considers that jaundice may not always occur in the course of the disease.

¹ "Liver Disease"; consulting editor, Sheila Sherlock, M.D., F.R.C.P.; editor for the Ciba Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch.; 1951. London: J. and A. Churchill, Limited. 8" x 5", pp. 262, with 112 illustrations. Price: 25s.

Stokes has endeavoured to show that the virus of infectious hepatitis will grow in the developing chick embryo, but so far his findings are not conclusive. A classical attack of hepatitis with jaundice has not been produced in the human volunteers after injection of "infected" chick embryo material.

There was much debate on the cause and control of ascites. Kunkel and Eisenmenger, of the Rockefeller Institute, New York, described their classical experiments on the dominant role of salt retention in the production of ascites in alcoholic cirrhosis. Physicians will do well to consider the wisdom of ordering a diet with a low salt content for these patients. However, they must not overlook the value of a diet rich in protein and vitamins.

And finally, A. I. S. Macpherson, who had enjoyed the privilege of working with both Blakemore in New York and Learmonth in Edinburgh, described the indications for the surgical relief of portal hypertension. He stated: "The aim of the surgical treatment is twofold—first to attempt to reduce the portal pressure; secondly, and possibly more important, to divert the blood from the dangerous cardioesophageal area." The value of the various surgical procedures remains the subject of vigorous controversy.

It is hoped that the Ciba Foundation may produce a further excellent symposium on liver disease in the near future.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Body Dynamics", by Eleanor Metheny, Ph.D.; First Edition; 1952. New York: McGraw-Hill Book Company, Incorporated. 9 $\frac{1}{2}$ " x 6", pp. 234, with 55 text figures.

Intended for the general reader who is interested in improving his own physical efficiency.

"The Early History of the Royal Alexandra Hospital for Children, Sydney, 1880 to 1905", by P. L. Hipeley, M.D. (Sydney), F.R.A.C.S.; 1952. Sydney: Angus and Robertson. 9 $\frac{1}{2}$ " x 6", pp. 120, with 37 illustrations. Price: 25s.

Contains a wealth of information about those who founded the hospital and fostered it in its early days.

"Brain Mechanisms in Coronary Disease: Causation, Treatment and Prevention", by N. E. Ischglondsky, M.D., with two appendices: 1. "From the Conditioned Reflex to the Science of Brain Dynamics." Paper delivered at the XVIIIth International Congress of Physiology. 2. "Reflexologic Basis of Personality." Paper delivered at the Ninety-Sixth Annual Meeting of the American Psychiatric Association; 1952. London: Henry Kimpton. 10" x 6 $\frac{1}{2}$ ", pp. 186, with 45 illustrations. Price: 25s.

The author is concerned with the science of brain dynamics.

"Practical Procedures", edited by Heneage Ogilvie, K.B.E., D.M., M.Ch., F.R.C.S., and William A. R. Thomson, M.D.; Second Edition; 1952. Published on behalf of *The Practitioner*. London: Eyre and Spottiswoode. 9" x 6", pp. 380, with 99 illustrations. Price: 25s.

Comprises twenty-four chapters on a variety of procedures by many authors.

"Malaria: Basic Principles Briefly Stated", by Paul F. Russell, M.D., M.P.H.; 1952. Oxford: Blackwell Scientific Publications. 9" x 6", pp. 222, with 64 illustrations. Price: 35s.

A monograph in "American Lectures in Infectious Agents and Disease".

"A Dictionary of Psychological Terms: With Definitions and Explanations", compiled by R. Macdonald Ladell, M.B., Ch.B.; 1951. London: The Psychologist Magazine. 7 $\frac{1}{2}$ " x 5", pp. 48. Price: 1s. 6d.

One of the "Practical Psychology Handbooks" intended for the general public.

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The Medical Journal of Australia

SATURDAY, JUNE 14, 1952.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

TUBERCULOSIS IN TASMANIA.

On September 24, 1949, attention was directed in these columns to a new *Tuberculosis Act* which had been put on the Statute Book of Tasmania. This Act made tuberculosis a notifiable disease and medical practitioners had to report any person who "is or may be" suffering from tuberculosis, or who was revealed by post-mortem examination to have been suffering from tuberculosis at the time of his death. In the notification, the practitioner was required to state whether the patient's disease was in an active form, or whether he was in an infectious condition. Under the Act, the Minister was given certain powers of compulsion. The Minister could require persons over fourteen years of age to undergo radiological examination of their lungs. If a person refused or failed to undergo examination, the director might nominate a medical practitioner to make the examination at a special place approved for the purpose. In certain circumstances, the director could order a patient to be removed to an institution "where he can be properly attended and treated" and detained for a period not exceeding six months. Among the provisions of the Act was one which stated that any person who, without lawful excuse, disobeyed any notice or order under the Act, would be guilty of an offence and the penalty would be £50 or imprisonment for three months. We described this Act as a piece of experimental legislation, because of the fact that it was to expire on June 30, 1950. In a letter to the journal a few weeks later, the Minister for Health of Tasmania, Dr. R. J. D. Turnbull, agreed that the Act was experimental, but stated that it was not because of its expiry on June 30, 1950. He wrote that the date had been inserted solely to try to move the Commonwealth Government to grant higher pensions. Opportunity has been provided for some insight into the working of this Act in the reports of the Minister for Health on the activities of his department for the years ended June 30, 1950, and June 30, 1951. It is, of course, clear that the Act has not expired.

The section on tuberculosis in the first report has been written by Dr. T. H. Goddard, who was Director of Tuberculosis; the section on tuberculosis in the second report has been written by Dr. James Tremayne, who succeeded Dr. Goddard in the directorship of the Tuberculosis Division. The claim is made (and it appears to be justified) that the compulsory X-ray provisions of the *Tuberculosis Act* have been responsible for an increased number of notifications of tuberculosis. During the first period, a total of 210 new cases of tuberculosis were notified. Of these infections 188 were pulmonary and 22 were non-pulmonary. Of the infections notified during the year, approximately two-thirds were shown to be in a moderately advanced, or in an advanced, stage. Of 121 such cases, approximately 50 occurred in persons residing in Hobart and adjacent areas, where compulsory X-ray examinations had been operating. This examination was regarded as having been instrumental in the discovery of these infections. In the whole series, mass X-ray examination and clinics were responsible for the discovery of 74 cases, and a further 70 were notified through general hospitals. This, it is stated, proves the benefit of mass X-ray examination, of the examination of contacts by clinics, and of the routine examination of patients in general hospitals. During the second period 236 cases of tuberculosis were notified. Of these, 210 were pulmonary. Included in the 210 patients with pulmonary tuberculosis were 35 who were eligible for treatment and benefits under the *Repatriation Act*. It is shown that 125 of the cases notified (59.5%) came within the twenty to twenty-four years age group, an age group covered by the compulsory X-ray provisions of the Act. In this group, females predominated over males. Of the 37 patients who were notified as suffering from advanced disease, approximately one-third were resident in the Hobart and adjacent areas, where compulsory X-ray examination was operative. In the Hobart and adjacent areas, the number of pulmonary notifications increased by 42 as compared with the figures for 1948, when X-ray examination was purely on a voluntary basis. The view is expressed that the routine chest examination of all patients admitted to public hospitals is an important means of discovering unsuspected cases of tuberculosis. It also provides a protection for the nursing staff. It is hoped that in the near future an X-ray plant will be installed at the Royal Hobart Hospital, so that the routine chest examination will be extended to cover all out-patients at the hospital. There is no doubt that it would be useful if this form of routine examination could be adopted in other hospitals throughout the Commonwealth. The Federal Council, at its last meeting, missed the opportunity to do something towards the extension of this form of examination. It received from the Director-General of Health for the Commonwealth a letter enclosing a communication from the Minnesota State Radiological Society in regard to the institution of radiological examination of the chests of all patients admitted to hospital, as a routine measure. Dr. J. B. G. Muir, of Hobart, pointed out on that occasion that the type of examination in question had been carried out at the Royal Hobart Hospital for about twelve months. The Federal Council adopted a pious resolution approving in principle of the procedure recommended by the Minnesota State Radiological Society. This approval in principle was not enough; all it did was to

consign the subject to decent oblivion. The matter should have been referred to the State Branches for their consideration and action.

Among the other developments which had taken place during the second period under review must be mentioned the establishment of a B.C.G. clinic in Hobart "for the vaccination of those groups of persons in the community at greatest risk, as well as others who work to avail themselves of this protection". Vaccination of all new entrants to the nursing staffs of the Hobart, Launceston and Devon hospitals has been continued, and this protection has been extended at the Launceston and Devonport chest clinics and at the Hobart B.C.G. clinic to contacts of tuberculous patients and children born into tuberculous households. It looks as if the Tasmanian Department of Public Health has been able to convince the Director-General of Health at Canberra that its members are capable of giving intradermal injections. (See *THE MEDICAL JOURNAL OF AUSTRALIA*, March 29, 1952, page 452.) Another step which has been taken is that examinations by the Mobile X-Ray Unit are no longer voluntary; the element of compulsion provided in the *Tuberculosis Act* has been introduced. Reference is also made by Dr. Tremayne to the introduction in July, 1950, of the Commonwealth Government's scheme of allowances for tuberculosis patients. The opinion is expressed that this scheme of pensions is the most effective system extant. Whether that statement is completely true or not does not really matter. The point is that reasonable provision is now made for sufferers from tuberculosis. And this must be looked on as due in no small measure to the repeated representations made by the Federal Council to the Minister over recent years. It would appear also that the Tasmanian Minister's intention to stimulate Commonwealth authorities by the declaration that the *Tuberculosis Act* of Tasmania was to last only twelve months, had some effect. It should be noted that under the heading of "projected undertakings" mention is made of the erection of a new chest hospital in southern Tasmania, and also that plans have been made for the provision of additional accommodation at the Northern Chest Hospital. Tasmania seems to have discovered the secret of occasionally getting things done, and we can expect that these plans will be put into effect. The next report on anti-tuberculosis measures in Tasmania will be awaited with interest.

Current Comment.

MEDICO-LEGAL ASPECTS OF PULMONARY EMBOLISM.

THERE are numbers of pitfalls in the clinical diagnosis of myocardial infarction, and the difficulties are not by any means removed in every instance by an appeal to the electrocardiograph. One feature of the comparatively recent expansion in knowledge of the graphic methods of examining the heart has been the similarity of the records of myocardial infarction and certain other conditions extraneous to the heart. Amongst these imitators are biochemical changes in the body fluids, especially their content of electrolytes, and pulmonary embolism. It used to be said that an electrocardiogram amongst other virtues could enlighten the clinician whether a cardio-vascular emergency suggesting an acute intrathoracic lesion was

due to coronary occlusion or pulmonary embolism. Now, although much greater precision is possible in the interpretation of electrocardiograms, records of a lesion of the acute *cor pulmonale* type may cause doubt to arise in the mind of even very experienced observers. Samuel A. Levine and J. P. Vazifdar have recently drawn attention to this fact with special reference to its medico-legal aspects.¹ Their communication is based on two cases which happened a few years ago; the facts were strikingly similar, and the electrocardiographic evidence on which they depended was misleading. The first patient, a man of thirty-seven years of age, sustained an injury of one leg, which necessitated strapping, and later the application of a cast in hospital. Two weeks later he had severe pain in the upper abdominal region, with a low-grade fever, and an electrocardiogram showed early take-off of *S-T1*, with depressed *S-T3*, and elevated *S-T* in lead *CF4*, changes which were thought to be characteristic of acute anterior coronary artery occlusion. He died some days later. One of the authors was asked for an opinion some two months later, and advised that, as the question of compensation was involved, an autopsy was advisable. Even at this late date there was clear evidence of thrombosis of the femoral vein, with adhesion of the clot to the vessel wall, and also thrombosis of several medium-sized arteries in one lung. There was no evidence of infarction of the heart or of disease of the coronary arteries. This new evidence made the justice of compensation apparent, as the sequence of events was now clear. The second case was that of a man, aged forty-five years, who suffered a fracture of one leg with severe crushing. He was ill for five months, and had considerable pain, relieved by a plaster casing or arthrodesis of the ankle. After a period of deterioration he died twenty-four hours after the onset of severe pain in the chest. Shortly before death an electrocardiogram gave evidence thought to be diagnostic of acute anterior coronary thrombosis. The later history was almost identical with that of the previous case, and here too an autopsy performed three months after death showed thrombosis in the veins of the injured limb, and in the pulmonary vessels also. The authors comment that the differential diagnosis between acute myocardial infarction and pulmonary embolism may be quite difficult. It will be noted that in each instance the diagnosis was considered to be plain, and there was thought to be no need for a post-mortem examination. All medical witnesses who have had to undergo cross-examination in cases in which the relation of injury to subsequent disability or death is at issue will surely agree that it is difficult to help the cause of justice when speculation or even probability and not irrefutable fact is the basis of argument. The real moral of this report is that post-mortem examination in such cases should be a matter of routine.

"CHLOROMYCETIN" INEFFECTUAL IN MUMPS.

IN *The Lancet* in 1950 a report was presented by P. Chalioungui to the effect that chloramphenicol had been effective in four cases in the treatment of mumps. This report was read by G. Nickerson and E. M. Warden in Montreal, and they decided to use "Chloromycetin" (the proprietary form of chloramphenicol) to give relief to two patients who were suffering from mumps orchitis.² No benefit resulted. As it was thought that these patients might have been exceptional in their lack of response, it was decided to undertake an investigation to see whether or not "Chloromycetin" would affect the course of mumps and prevent any of its complications. The plan of treatment was to give "Chloromycetin" in doses of 2.5 grammes per square metre of body surface—that is, about 4.0 grammes for an adult in four equal daily doses for five days—and to observe the effects. The series of patients included 57 consecutive children, aged one to fifteen years, who were suffering from mumps uncomplicated by other

¹ *The American Journal of the Medical Sciences*, December, 1951.

² *The Canadian Medical Association Journal*, January, 1952.

diseases. The "Chloromycetin" was found to be totally without any effect on the course of the disease. "Chloromycetin" failed to have any effect on three patients who suffered from associated orchitis, and a fourth patient developed orchitis on the fourth day of "Chloromycetin" administration. Four females who had swelling of the salivary glands, but who were afebrile, developed fever and signs of meningeal irritation during "Chloromycetin" administration. Examination of their cerebro-spinal fluid revealed findings consistent with a diagnosis of mumps meningitis.

This negative report is of some value, especially in these days, which are full of therapeutic surprises. Aureomycin has been reported to be of value in mumps orchitis.¹ If the report of Nickerson and Warden does no more than curb the exuberance of some therapeutic optimist, it will not have been made in vain.

RADIOPHOSPHORUS (P^{32}) IN DIAGNOSIS AND TREATMENT.

RADIOPHOSPHORUS (P^{32}) was the first radioactive isotope to be used in experimental medicine in 1935. Since then it has been used extensively both for diagnosis and for treatment. A useful summary of the results obtained by its use has been given by J. Duffy, junior, and J. W. Howland.² Radiophosphorus (P^{32}) has a half-life of 14.3 days. It emits only β rays, which have a very short range in tissues, namely, some three to four millimetres. It is used as a solution of disodium hydrogen phosphate with a specific activity of five to seven millicuries per millilitre. The amount of irradiation from one millicurie of P^{32} in a 70-kilogram adult is equivalent to 0.6r of total body irradiation for the first day or 12r during the complete decay of the P^{32} , if we presume no excretion of P^{32} . This amount of whole body irradiation is proportional to the dosage of external irradiation which is given in the treatment of blood diseases. It is rapidly absorbed from the intestinal tract. Approximately 20% to 40% of an orally administered dose may be excreted in the faeces during the first four to six days, but this may be kept at a minimum by administration to the patient in the fasting state. Approximately 5% to 25% of an intravenous dose is excreted, mostly in the urine. After administration, either oral or intravenous, P^{32} initially becomes localized in erythrocytes with an increasing concentration in leucocytes after forty-eight hours. This is most pronounced in leucæmic blood, the concentration of P^{32} in leucocytes being four to five times the amount in erythrocytes one week after administration. There is some evidence of increased uptake in the more rapidly growing tumours, but as bone-marrow concentration is always above any tumour-concentration, treatment with P^{32} must be limited to the blood diseases. It must be considered a general cell poison. *Polycythæmia vera* and chronic leucæmia are the only conditions in which P^{32} treatment may be definitely indicated. For *polycythæmia vera* the oral administration of P^{32} has been shown to be a safe, convenient and practical method of therapy. The initial dose of P^{32} depends on the initial number of erythrocytes. Additional doses are given at intervals of not less than two months. Radioactive phosphorus does not destroy already formed erythrocytes but inhibits further production of red cells. Since specific prolongation of life with P^{32} is not demonstrable, secondary factors must decide whether the treatment is worth while. After treatment with P^{32} patients often die of acute leucæmia, but there is no evidence of a real increase in the incidence of leucæmia in polycythæmics. A definite prolongation of life has been observed in patients with chronic leucæmia treated with P^{32} over those treated with X rays alone. It is unnecessary and dangerous to attempt to depress a leucæmic count to normal limits with P^{32} . No benefit has been observed in the treatment of acute

leucæmia. A great deal of work has been done on the uptake of P^{32} by neoplastic tissue in cancer diagnostic studies. Growing cancer cells show a higher content of P^{32} than surrounding normal cells, but one limitation in the diagnosis of any but superficial growths is the short β range of the P^{32} rays.

Red cells labelled with P^{32} have been used for the determination of total blood volume. On incubation of isotonic sodium phosphate with blood drawn from the patient, over 40% of the labelled phosphate will enter and label the red blood cells. This blood is then reinjected into the patient and the blood volume determined by the dilution technique. The method has been shown to be the most accurate relatively simple method of blood volume determination available if the necessary apparatus is at hand. Circulation time can be determined by injecting isotonic saline containing P^{32} into a cubital vein with the patient in the recumbent position. A Geiger counter on the sole of the foot indicates the arrival of the P^{32} at the foot.

While the usefulness of radioactive phosphorus is disappointingly small in clinical medicine, it has great value in investigations of specific metabolic problems because of the wide distribution of phosphorus in cell nucleoproteins and other cell constituents.

A DISINFECTANT BARRIER IN BURN DRESSINGS.

LEONARD COLEBROOK AND A. M. HOOD in 1948 reported experiments which showed the ready passage of organisms through dressings soaked with sterile plasma plus broth,¹ and it appears that not only may burns and surgical wounds be infected from the exterior by this means, but also organisms may "grow through" from an infected burn to the exterior and form on the surface of the dressing a reservoir from which cross-infection may take place. Plastics, such as "Cellophane" and "Nylon" derivatives, act as a barrier, but they do not allow full evaporation from the surface and are difficult to apply in certain sites. Another possible way of overcoming the problem has now been suggested by E. J. L. Lowbury and A. M. Hood.² They found experimentally that cultures of *Pseudomonas pyocyanus*, *Proteus vulgaris*, *Bacterium aerogenes*, *Staphylococcus aureus* and *Streptococcus pyogenes* would not "grow through" model dressings soaked with sterile serum-broth when the cotton wool was medicated with phenyl-mercuric bromide. Dry treated dressings dipped in serum-broth cultures of bacteria did not prevent the passage of the organisms, but the surface of the dressing became sterile in periods varying from one to six hours or within twenty-four hours. Treatment of cotton wool with other phenyl-mercuric salts or with octyl cresol and treatment of crêpe bandage with phenyl-mercuric bromide did not provide a reliable disinfectant barrier. From their experiments Lowbury and Hood conclude that it may be possible to prevent the contamination of sterile burns through soaked dressings by using a layer of cotton wool treated with phenyl-mercuric bromide and so avoid the disadvantages of plastic materials. They consider that the value of the treated dressings as a barrier to the passage of organisms from an infected burn through soaked dressings is more doubtful, and suggest that the experimental conditions may not reproduce difficulties presented by a burn which continues to exude serum after the dressing has become soaked through. In either case the clinical significance of the work remains to be determined. Lowbury and Hood state that in the burns unit at the Birmingham Accident Hospital, where they work, dressings are replaced as soon as they become soaked through with exudate. However, in the event of a burning catastrophe, or in other circumstances in which dressings cannot be changed at short notice, it may be valuable to incorporate a disinfectant layer in the cover applied to burns as a routine. Results of a clinical trial along these lines would be of great interest.

¹ M. P. R. Spinelli, N. L. Cressy and P. Kunkel, *Connecticut Medical Journal*, 1951, Volume XV, page 43; quoted in "The 1951 Year Book of Drug Therapy".

² *New York State Journal of Medicine*, March 1, 1952.

¹ *The Lancet*, October 30, 1948.

² *Ibidem*, May 3, 1952.

Abstracts from Medical Literature.

THERAPEUTICS.

Treatment of Vesiculocerosive Stomatitis with Aureomycin Troches.

A. A. FISHER AND M. LEIDER (*Archives of Dermatology and Syphilology*, August, 1951) state that lesions anywhere on the mucous membranes present problems both of etiology and of therapy. Mechanical trauma, burns, primary chemical irritation, bacterial, fungous and virus infection, and drug and contact eruptions, among others, are all possibilities in most cases. The authors report their favourable experience with the treatment of recurrent non-febrile aphthous stomatitis with aureomycin in the form of troches in eight cases of vesiculocerosive stomatitis. There were complications from the therapy in more than half the cases—mild diarrhoea, *pruritus ani* and *pruritus ani et vulva*. The conditions ceased of themselves. Two patients acquired a new form of stomatitis. The authors state that this type of stomatitis is apparently common after topical treatment with antibiotics, particularly after the use of aureomycin and penicillin. It shows itself as a bright redness of the entire oral cavity, beefy glossitis, *perleche* and chelitis. The cause of "aureomycin mouth" is not clear, but most evidence suggests that it is the result of ariboflavinoic acid, and that adequate doses of the riboflavin fraction of the vitamin B complex are rectifying.

Hyaluronidase in Local Anesthesia.

D. C. MOORE (*Anesthesiology*, September, 1951) presents a large series of cases which he has examined to evaluate the use of hyaluronidase in local anesthesia. He states that fascia acts as a barrier to the enzyme. If adrenaline is added to the local anesthetic agent, hyaluronidase does not affect the analgesia time, nor does the adrenaline affect the diffusion caused by the hyaluronidase. Onset of anesthesia is quicker, and smaller volumes of solution are necessary. Allergic reactions may occur, and toxic reactions to the local anesthetic agents and vasopressors are more likely. Hyaluronidase was found helpful in procedures involving infiltration of tissues, but the increase of successful regional blocks with its use is doubtful.

Surgical Anesthesia and the Heart.

HARRY GROSS *et alii* (*Current Researches in Anesthesia and Analgesia*, September-October, 1951), in a paper on surgical anesthesia, heart disease and cardio-vascular responses, stress the importance of thorough investigation and treatment of the cardiac patient by the physician and anesthetist before, during and after operation. Special care should be taken with the choice of time of operation and the posture during operation. Choice of the anesthetic drug, the avoidance of excitement with the production of adrenaline, anoxia, cardiac toxicity, and post-operative vomiting should be considered. Ether is favoured

for lack of cardiac toxicity or tendency to cause anoxia. Cyclopropane is explosive and very prone to produce cardiac irregularities at any level of anesthesia except when used with ether. Nitrous oxide is not satisfactory without supplement. Intravenously administered barbiturates are useful for short procedures and as adjuvants for other general and regional anesthetics. Spinal anesthesia is favoured, except for hypertensives, provided adequate measures are taken to maintain normal blood pressure. The authors suggest the use of spinal anesthesia as a bloodless phlebotomy in the management of acute left ventricular failure. The use of "Avertin", in a dose of 60 to 80 milligrams per kilogram, is recommended in cases of heart diseases, especially for the thyrotoxic patient. Curare is recommended for long intraabdominal operations, renal disease being a contraindication. The authors state that cardiac arrhythmias may be caused by anoxia, psychogenic factors, such as pain, tracheal intubation and irritant gases, particularly cyclopropane. They deny that vagal reflexes are important in the production of cyclopropane-adrenaline arrhythmias. They state that impulses travel by visceral afferent fibres through the celiac and superior mesenteric plexuses, splanchnics and spinal cord to a brain centre above the pons, probably the hypothalamus. Efferent impulses then pass to the heart by way of the cardiac sympathetics and increase the irritability of the heart. The theory of causation of cardiac failure following pulmonary embolism is discussed.

Relief of Pain.

H. K. BEECHER (*Anesthesiology*, September, 1951) discusses the problem of pain and its alleviation. His study concerns the relief of post-operative pain with morphine sulphate, pentobarbitone and saline. Four categories of response were observed: (i) no comfort, no pain relief; (ii) no comfort, pain relief; (iii) comfort, no pain relief; (iv) comfort, pain relief. When pentobarbitone was used, the analgesia was less than with morphine, but the hypnosis was greater. Half of the patients while under the effects of pentobarbitone either did not experience what is commonly called pain or were not made uncomfortable by it.

Methylphenylsuccinide and Petit Mal Epilepsy.

FREDERIC T. ZIMMERMAN (*Archives of Neurology and Psychiatry*, August, 1951) states that treatment of *petit mal* seizures is often inadequate. He considers methylphenylsuccinide equal to, if not superior to, trimethadione, and it is relatively non-toxic. The average daily dose is 2.4 grains in units of 0.3 grain capsules. Toxic signs are few, but if they occur they are in the following order of frequency: nausea, dizziness, drowsiness, vomiting, headaches, and dream-like states.

Terramycin and Syphilis.

C. LEVADITI AND A. VAISMAN (*Journal of Antibiotics and Chemotherapy*, October, 1951) describe terramycin as an antisyphilitic. They state that penicillin was effective in syphilis, but chloramphenicol and aureomycin were not so to the same degree. Terramycin has been shown to be effective against primary and secondary lesions. Oral

treatment was satisfactory. For their investigations the authors used rabbits. Intramuscularly administered terramycin, 100 milligrams per kilogram daily for three days, caused immobilization of treponemata, their disappearance on the third day, healing of lesions on the fourth day, and sterilization of the blood and peripheral lymphatic system on the forty-fifth day. Larger doses were too toxic. Mice were affected in the same way as rabbits, but the authors consider that further study is necessary for use of the treatment for human subjects.

Methyl Gallate.

H. J. CARLSON *et alii* (*Journal of Antibiotics and Chemotherapy*, October, 1951) describe the properties of methyl gallate isolated from *Koelreuteria paniculata*, an Asian tree of the Sapindaceae family. This antibiotic, methyl gallate, was isolated and purified. The substance was found to inhibit the growth of Gram-positive and Gram-negative bacteria and fungi. Crude and pure fractions of the plant inhibited influenza virus.

Sensitivity to Antibiotics.

J. C. MONNIER AND E. B. SCHOENBACH (*Journal of Antibiotics and Chemotherapy*, October, 1951) discuss sensitivity of bacteria to various antibiotics. They state that 12 strains of organisms were tested. Resistance to streptomycin occurred with all organisms. Aureomycin and terramycin gave rise to resistance in staphylococci and Gram-negative bacilli, but not in streptococci. Tolerance to chloramphenicol was demonstrated with Gram-negative bacilli, but not with Gram-positive cocci. Resistance to penicillin of moderate degree could be produced temporarily in all Gram-positive cocci. Established. If resistance developed to aureomycin, a change to terramycin was not of value, but penicillin, chloramphenicol or streptomycin might be used effectively.

Cortisone and ACTH.

S. W. COSCRIFF (*The Journal of the American Medical Association*, November 3, 1951) describes thrombo-embolic phenomena associated with cortisone and ACTH therapy. Of 700 patients reviewed, 28 developed 40 episodes of thrombo-embolic disease. These resulted from hypercoagulability of the blood associated frequently with these agents. The age of the patients affected was about the usual age for such complications; 75% were between forty and seventy-nine years of age. The diseases under treatment were varied, comprising most of those most commonly treated with cortisone and ACTH, such as pemphigus, rheumatoid arthritis, dermatitis, scleroderma and the like. Doses of cortisone were 50 to 100 milligrams daily, and of ACTH 40 to 80 milligrams daily. The higher doses appeared to produce more of these complications, which were fairly equally divided between cortisone and ACTH.

Aureomycin.

H. D. BRAINHERD *et alii* (*Journal of Antibiotics and Chemotherapy*, October, 1951) describe studies on aureomycin. They state that aureomycin was detected in the serum in one hour after administration of 250 milligrams to

1.5 grammes by mouth. After 250 milligrammes the peak in the serum was at two hours, declining at four and six hours to negligible amounts in twenty-four hours. After administration of one grammme of aureomycin, peak concentrations in the serum were at four hours. Intramuscular injection was far less effective. In the spinal fluid the concentration was poor, as it was in the pleura. Aureomycin was detected in joint fluid, bile and urine—the last-mentioned particularly. In the faeces it was strongly concentrated. Nausea and vomiting occurred as the daily dose increased. Administration of 250 milligrammes every six hours caused some nausea and loose stools. Rashes and vertigo were noted. No toxic effects on kidneys, liver or blood were observed. Oral administration of aureomycin was most effective; 250 milligrammes were given every six hours. The drug was active against most bacteria which prevailed, except *Pseudomonas aeruginosa* and *Proteus vulgaris*.

NEUROLOGY AND PSYCHIATRY.

Contested Wills.

W. G. ELIASBERG (*The Journal of Nervous and Mental Disease*, April, 1951) discusses the reasons for contesting wills and deals particularly with the problems of handwriting and the validity of handwriting experts. He states that the number of errors committed in this field have been numerous owing to insufficient training and inherent difficulty. He refers to problems arising from the fact that the moving hand of the writer was held and supported. He also gives a case history of a man with partial competency in an aphasic defect, who was able to conceal that he had agnosia relating to figures; whilst quite competent to make decisions on general principles of his will, the man was quite incompetent to make decisions in relation to the amount of money involved. The author has developed a test for such patients, which he is willing to send to any inquirer. He stresses the view that psychiatric examination at the time of making wills is important in many "twilight states" including anoxæmia and epilepsy.

Electrocardiography and Emotion.

GASTON E. BLOM (*The Journal of Nervous and Mental Disease*, April, 1951) reviews electrocardiographic changes in emotional states, commencing with the observation of Hyde and Scalapino in 1918 that music could alter the *R* wave in lead II. He discusses the changes produced by drugs, exercise, hypoglycæmia and hyperventilation. He states that the literature suggests that changes due to emotion may be produced by a variety of mechanisms. The most common is by impulses over the vagus and accelerator nerves to the heart. The author records a series of pre-shock electrocardiograms from 193 acutely psychotic patients from the Navy and Marine Corps; 93 (42%) are abnormal. The abnormalities have been tabulated. The most striking findings were the high incidence of *S-T* segment and *T* wave abnormalities, which were largely of sympathetic type, low *T* waves and *S-T* depression. The author infers a

more predominant sympathetic effect on the ventricular portion of the electrocardiogram with a more parasympathetic effect on the auricular portion.

Boy Heroin Addicts.

P. ZIMMERING *et alii* (*The Journal of Nervous and Mental Disease*, July, 1951) discuss heroin addiction in adolescent boys. They state that there has been a sudden widespread use of heroin among boys of New York City in the Harlem area. Twenty-two case records are discussed. All the boys except one were Negroes or of Puerto Rican descent. The authors state that the habit usually commences through curiosity, which is stimulated by a peddler. The boy begins by sniffing (or "snorting") through his nose. He may then switch to the subcutaneous route ("skin popping"), and finally uses the intravenous route ("shooting the main line"). The latter procedure is usually accomplished by fitting a hypodermic needle to an eye dropper and injecting a solution of heroin after heating in a spoon over a flame. The sequence of reactions includes a lessening of tension, a feeling of adequacy, definite euphoria and a sensation of flying through space. Afterwards there is a feeling of depression. Once the addiction is complete there is difficulty in breaking it off. Crimes and delinquencies are almost inevitable in order to provide money for more capsules. The authors stress certain psychological factors, which include a feeling of social inferiority, a close and emphatic relationship with their mothers, weak object relationship with others, omnipotent strivings and a tendency to regression. They believe that the problem is one for social psychology and the police. Youthful addicts should be sent to institutions for "normal" boys for periods up to two or three years with periodic trials at home.

Tumours of the Spinal Cord with Choked Optic Disks.

J. GRAFTON LOVE, HENRY P. WAGNER AND HENRY W. WOLTMAN (*A.M.A. Archives of Neurology and Psychiatry*, August, 1951) state that bilateral papilledema has been reported in cases of tumours of the cervical part of the cord, particularly if near the *foramen magnum*. Some cases of papilledema have been described in patients with upper thoracic tumours. However, the authors report two cases in which choked optic disks occurred in patients with tumours low in the spinal cord. They state that the cause of the papilledema is obscure. These cases are so rare that it is usual to consider association of a spinal cord lesion with papilledema to indicate both intracranial and spinal cord lesions.

Electroshock and Schizophrenia.

D. M. PALMER, H. E. SPRANG AND C. L. HANS (*The Journal of Nervous and Mental Disease*, August, 1951) discuss electroshock therapy for schizophrenia based on its application to 455 male veterans; 198 (43%) showed either great or moderate improvement. The authors point out that the remission rate, 17.6%, after five months to forty-two months from the time of treatment is not remarkable. They conclude that the greatest value of electroshock in this condition is its usefulness in getting the patient into a suitable state

for other forms of treatment, such as psychotherapy and "milieu" therapy. Furthermore results cannot be gauged on hospital discharge rate alone, as many patients who do not leave hospital are improved in relation to eating and hygiene.

Electroconvulsive Therapy and Conservative Treatment in Depressive States.

ELIOT T. O. SLATER (*The Journal of Mental Science*, July, 1951) states that the statistical data provided by Karagulla in a paper published in October, 1950, in *The Journal of Mental Science* do not justify two of her main conclusions, which were that electric convulsion therapy is without effect either on the recovery rate or on the duration of stay in hospital in depressive states. A more careful examination of the data suggests that the exact opposite of both propositions is correct.

Nervous and Mental Disorders in Cushing's Syndrome.

JOHN D. SPILLANE (*Brain: A Journal of Neurology*, Volume LXXIV, Part I) reports on clinical features in seven cases of Cushing's syndrome. One patient showed improvement after irradiation, but four years later was blind, bedridden and incontinent. Two patients were psychotic in the early stages of the illness. Two others were psychoneurotic in the early stages. One had considerable depression and alteration of personality. The author states that published case histories show that mental disorders are frequent in Cushing's syndrome.

Emotion and Cardiac Disease.

MORTON F. REISER (*The American Journal of Psychiatry*, April, 1951) discusses the emotional aspects of cardiac disease. He quotes personal records of 25 consecutive patients with congestive heart failure. In 19 cases emotional factors played a predominant role in precipitation. The author stresses the complicated pattern of psychological sequences. The patient must integrate new ideas of his own limitations, and he must work out his previous psychological outlook. The onset of cardiac disease is a major form of psychological trauma. The key to its successful therapy lies in the doctor-patient relationship. Most important is the handling of the anxiety and fears of heart disease. The role of the psychiatrist in such cases is to help the physician to deal with the emotional problem, to determine if major psychotherapy is needed and to deal with the physician's own anxiety where indicated.

Polyneuritis following Cutaneous Diphtheria.

JOHN A. DI FIORA (*The Journal of Nervous and Mental Disease*, October, 1951) describes a small group of servicemen in whom various types of polyneuritis presented similar cutaneous scars. In nine cases, all but one had lesions of lower extremities. The scars have depressed centres and peripheral hyperpigmented borders. It is inferred that the acute sores occur by direct transmission as in wiping of the nose or mouth. The skin may be abraded by "jungle rot" or insect bite, for example, by lice. In all cases the patients improved to complete recovery.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XXIV.

THE CLINICAL USE OF THE SULPHONAMIDES.

IN 1935 Domagk introduced sulphonamidochrysoidin ("Prontosil") for the treatment of streptococcal infections. Sulphanilamide was soon proved to be its active component, but many substitution products with a wider range of therapeutic activity rapidly replaced this drug. Acetylation in the sulphonamide group yielded sulphacetamide, now used chiefly for instillation in the eyes in conjunctivitis as aqueous solutions of 10% to 30% strength.

Substitution in this same group with pyrimidine and its mono-methyl and di-methyl derivatives gave sulphadiazine, sulphamerazine and sulphamezathine, the compounds now most commonly used in systemic infections. These three drugs are of low toxicity with practically identical therapeutic effects. They are more slowly excreted than the other sulphonamides, sulphamerazine being excreted most slowly, and so it has the advantage of requiring less frequent dosage. Sulphamezathine, in both its unaltered and acetylated forms, is more soluble than the other two and thus is less liable to cause urinary complications. Given orally, they are almost completely absorbed, but because of their relative insolubility, the more soluble sodium derivatives are used for intravenous injection when a rapid effect is desired. Great care is necessary to avoid leakage outside the vein since the considerable alkalinity of these compounds renders them injurious to the tissues. Mixtures of these sulphonamides in varying proportions and also mixtures of some of them with sulphathiazole have been used to avoid precipitation of their crystals in the urinary tract. "Sulphadit", "Sulphatriad" and "Dimerzine" are examples of such mixtures.

"Gantrisin" (3,4 dimethyl-5-sulphanilamidoisoxazole), available here relatively recently, has therapeutic activity about equal to sulphadiazine, with the added virtue of much greater solubility than sulphamezathine or the various sulphonamide mixtures.

The other important group of sulphonamides comprises those poorly absorbed from the bowel. First came sulphaquindine, later the still less readily absorbed succinylsulphathiazole and phthalylsulphathiazole, and lastly phthalylsulphacetamide. Poor absorption allows the use of large doses without risk of toxic effects and results in high concentration of the drugs in the bowel, hence their use in certain intestinal infections, especially the bacillary dysenteries, and in the preparation of patients for intestinal operations.

Related sulphone compounds (such as "Promin" and "Promizole") are used in the treatment of leprosy and have been used sometimes in addition to streptomycin and para-aminosalicylic acid in tuberculosis.

The Mode of Action of the Sulphonamides.

Sulphonamides are bacteriostatic, not bactericidal, preventing multiplication of susceptible organisms and so helping the natural defences of the body to eliminate the invaders. These drugs compete with para-aminobenzoic acid, an essential nutrient for susceptible organisms, and a substance which readily inhibits the action of sulphonamides. Sulphadiazine must be present in one hundred times the concentration of this acid to compete successfully with it. If bacterial multiplication is not completely prevented some organisms acquire resistance to high concentrations, not only of the particular drug used, but of all the sulphonamides. This is particularly shown by the gonococcus, though practically all susceptible organisms may develop resistance to these drugs. This is unlikely to occur with short, effective therapy, but may be important in prolonged prophylactic treatment. These considerations indicate the necessity for early treatment with adequate doses of these drugs.

Compounds with a chemical nucleus similar to para-aminobenzoic acid inhibit the action of sulphonamides. This is important with the use of certain local anaesthetics with this chemical structure, for example, procaine, benzocaine and butocaine; those without this structure and action include cocaine, "Stovaine", "Decicain" and "Nupercaine".

Yeasts, pus and necrotic tissue also inhibit the action of sulphonamides.

The Fate of the Sulphonamides in the Body.

After absorption, the sulphonamides are distributed evenly throughout the body and pass into the milk and across the placenta to the fetus. Maximal concentration in the blood is usually attained within four hours; hence doses at four-hourly intervals are generally necessary to maintain an adequate level of the drug in the blood. A variable, usually small, amount of the drug is destroyed in the body. Excretion occurs very largely through the kidneys, both in an unaltered form and as a less soluble acetylated derivative. Acetylation at the amino group takes place in the liver, producing a therapeutically useless but toxic compound. (In forming sulphacetamide, acetylation of sulphanilamide occurs in the sulphonamide group.)

Toxic Effects of the Sulphonamides.

When the urine is unduly concentrated, crystals of these substances may be precipitated in the renal tubules and cause renal colic, haematuria and diminution in urinary output even to the point of anuria. The use of sulphonamides is contraindicated in diseases in which the urinary output is appreciably reduced, for example, acute nephritis.

All sulphonamides and their acetylated derivatives are more soluble in alkaline than in acid or amphoteric urine; hence the urine should be kept alkaline by the use of potassium or sodium citrate and sodium bicarbonate. To prevent undue concentration of the urine, the fluid intake should exceed three litres (five pints) daily to ensure a urinary output above 1.5 litres (50 ounces). Both intake of fluids and output of urine should be charted. Should urinary complications arise from failure to take these precautions, they must be instituted immediately and the intake of the drug stopped. In the worst cases, ureteral catheterization with irrigation of the renal pelvis may restore the flow of urine. Such precautions are still more essential when increased loss of fluids occurs, as with diarrhoea, vomiting or excessive sweating. If the necessary fluid cannot be taken by mouth, it should be introduced by any available route, rectally, subcutaneously, intraperitoneally or intravenously. These measures should be continued for two days after stopping administration of the drug, since the commonly used sulphonamides are relatively slowly excreted. The use of sulphamezathine or the more soluble "Gantrisin" or mixtures of sulphonamides in appropriate cases reduces the risk of these complications.

Apart from these physical effects in the urinary tract, various other toxic results may be produced by the sulphonamides. The modern drugs are less toxic than the earlier ones and such manifestations as cyanosis and mental clouding are rarely seen today. Headache, anorexia, nausea and vomiting are still common, but are relatively unimportant. The chief toxic effects today are agranulocytosis, drug fever and drug rashes.

Agranulocytosis occurs from the eighth day onwards, with the usual symptoms of sore throat, fever and malaise. To reduce its incidence, the drug should be stopped immediately if the leucocyte count falls appreciably below 4000 per cubic millimetre or the percentage of polymorphonuclear cells falls abruptly. Other serious blood disorders such as purpura, aplastic anaemia and acute haemolytic anaemia are rare. The latter tends to appear early, in the first few days of treatment. With these complications the drug should be stopped immediately and the usual treatment for these disorders instituted. In agranulocytosis, antibiotics are given with ACTH or cortisone and perhaps also "Pent-nucleotide".

Drug fever is not uncommon, either during the treatment or within twenty-four hours of the stopping of therapy. It is rare before the second week unless these drugs have been used previously. A rising temperature during treatment is suggestive, while fever persisting after a week of treatment means therapeutic failure or drug fever and administration of the drug should be stopped. If the fever is due to the drug, it will then abate rapidly. A rise of temperature more than two days after treatment has ceased is certainly not due to the drug.

The usual skin lesion is a papulo-erythematous rash, associated with fever. If these drugs have been used previously, it may appear within a few hours of the first dose, otherwise it is not seen until after the first week of treatment. Rarely an exfoliative dermatitis occurs. Photosensitivity occasionally develops, hence exposure to direct sunlight or ultra-violet light should be avoided while these drugs are being taken.

In diseases due to hypersensitivity, the use of drugs known to be sensitizing agents should be avoided.

There is evidence that *polyarteritis nodosa* may rarely follow as a result of sensitization to the sulphonamides. These drugs are one potent factor in the increased incidence of the collagen diseases. It is unwise to give sulphonamides in chronic discoid *lupus erythematosus* and still more so in an acute case.

Direct toxic effects of these drugs may appear early, for example, gastro-intestinal symptoms, headache and acute haemolytic anaemia. Those appearing after the first week, drug fever, rashes and agranulocytosis (unless these drugs have been taken previously when they may appear rapidly) are attributed to sensitization to these compounds and anti-histamine drugs may be of help in their treatment. Because of these complications, the use of these drugs for more than one week is generally unwise and in any case is seldom useful. Sensitization is usually to the whole group and not to a specific sulphonamide. Before commencing treatment it is wise to inquire which sulphonamides, if any, have been taken previously and whether any toxic effects resulted.

Therapy with the Sulphonamides.

Organisms reasonably susceptible to the sulphonamides include, among others, Group A β haemolytic streptococci (responsible for most human streptococcal infections), pneumococci, meningococci, *Bacterium coli*, *Bacterium dysenteriae*, gonococci, staphylococci, Ducrey's bacillus, actinomycetes, *Clostridium welchii*, and the virus of *lymphogranuloma inguinale*. The introduction of the various antibiotics greatly reduced the use of sulphonamides, and in treatment of infections today their use is practically limited to the first five organisms mentioned above. Only in meningococcal and bacillary dysenteric infections are they better than the appropriate antibiotic, though their use in combination with antibiotics is valuable in many infections. They have some advantage in lower cost and ease of administration as compared with antibiotics.

The local application of sulphonamides to burns and other lesions of the skin is inadvisable because of the risk of sensitization of patients to these compounds, while other equally effective substances are available. The dusting of powdered sulphonamides into surgical wounds is often followed by the formation of granulomata of the foreign body type, with subsequent sinus formation. Aqueous solutions of sulphacetamide in concentrations up to 30% are extensively used as eye drops in appropriate superficial infections in the eyes and in the small dosage used carry little risk of sensitization.

The sulphonamides are usually available as tablets containing 0.5 gramme. They should be chewed or crushed up to aid absorption. For small children, the drug may be given in suspension or as specially flavoured tablets containing 0.25 gramme.

There are certain accepted principles in sulphonamide therapy. Treatment must be intensive, continuous and instituted as early as possible to combat the infection and prevent the development of drug-resistant strains of the organism. Hence a large initial or loading dose is followed by high dosage for three days, by medium doses for two days and by lower doses for the last two days. Treatment should continue for two or three days after the patient is afebrile, but the use of these drugs for more than a week is rarely advisable and greatly increases the risk of complications.

If after three days of full treatment there is failure to respond to the drug, the case should be reviewed very carefully, preferably with estimations of the level of the drug in the blood and of the sensitivity of the organism if the use of the drug is to be continued. Usually it is preferable to change to the appropriate antibiotic or to use this in addition to the sulphonamide. With the usual doses of sulphadiazine (given below) used in moderate and in very severe infections, the level of the drug in the blood varies from 6.0 to 15.0 milligrammes per 100 millilitres of blood.

Oral administration is the method of choice, but in very severe infections or with troublesome vomiting, a loading dose intravenously may be advisable and even repeated doses may be necessary occasionally, though administration by mouth or stomach tube should be utilized as soon as possible.

For the adult of average weight (about 10 stone) the dose of most sulphonamides in very severe systemic infections (such as meningococcal meningitis) should be a loading dose of from 2.0 to 4.0 grammes intravenously with 1.5 grammes by mouth (or 4.0 to 6.0 grammes orally), followed by maintenance doses of 1.5 grammes every four hours for three days, then 1.0 gramme four-hourly for two days, and then six-hourly for the last two days. For milder infections,

an initial oral dose of 2.0 grammes is followed by 1.0 gramme four-hourly for three days, and, if the response is satisfactory, this dose is given six-hourly until the temperature has been normal for two days. With sulphamerazine, because of its slow excretion, maintenance doses may be given at six instead of four hour intervals and, in milder infections, double the usual six-hourly dose may be given every twelve hours. This is the chief advantage of this drug, since sleep need not be interrupted to administer it.

Children, weight for weight, tolerate sulphonamides better than adults. Up to about the age of one year, the four-hourly dose may be 0.1 gramme per kilogram of body weight, with an initial dose of double this amount. For those of average weight between one and three years, one-third of the adult dose, from four to ten years, half the adult dose, and from eleven to fourteen years, two-thirds of the adult dose is given.

In infections of the urinary tract with susceptible organisms, somewhat smaller doses may suffice because the drug is concentrated greatly in the urine. The susceptibility of the organism is preferably determined before the commencement of treatment (though the result of the test need not be awaited), and this should be done if there is not an adequate response within three days, unless, as is usually advisable under these circumstances, a change to or the addition of the appropriate antibiotic is made. The more soluble "Gantrisin" is the sulphonamide of choice in these infections, and if the urine is made alkaline only a normal fluid intake is required. A greater concentration of the drug in the urine is thus achieved than if the fluids are forced. A dose of 6.0 grammes daily for a week should be sufficient to eradicate the susceptible organism in the absence of urinary obstruction or other pathological process, such as calculus, which may predispose to infection. Since these infections frequently relapse (if the patient is followed for some months) and since infection with more than one organism is common, the use of "Gantrisin" and the appropriate antibiotic or mixture of antibiotics is often preferable to the use of one alone. A relapsing infection demands full investigation of the urinary tract for predisposing or exciting causes.

The Use of Sulphonamides in Intestinal Diseases.

In intestinal infections, larger doses of the more poorly absorbed sulphonamides are usually given, though at least equally good results are claimed for the less expensive sulphadiazine, used in the same dosage as for systemic infections of moderate severity. But the negligible risk of toxic effects with the former group (except perhaps with sulphaguanidine) leads to their being generally preferred. In the bacillary dysentery a loading dose of 6.0 grammes of sulphaguanidine or succinylsulphathiazole is given, followed by maintenance doses of 3.0 grammes every four hours until the stools are reduced to four daily, when the same dose is given every eight hours until cure is achieved. Phthalylsulphathiazole may be given in half these doses. Since toxic effects are negligible, these drugs may be used for more than a week, though an adequate supplement of vitamins of the B group should then be provided, because of the reduced synthesis of these vitamins following alteration of the intestinal flora.

An occasional patient with non-specific ulcerative colitis benefits from the use of these drugs by mouth and as a retention enema of 8.0 to 10.0 grammes suspended in mucilage and given daily for ten days. They are extremely valuable in the preparation of patients for operations on the bowel. In these conditions, smaller doses are used than for the treatment of bacillary dysentery.

The Use of Sulphonamides in Prophylaxis.

The prophylactic use of sulphonamides in gonorrhoea and chancroid is frequently successful, but resistant strains develop more readily with the gonococcus than with other organisms and penicillin has proved more successful for this purpose.

In semiclosed or closed communities, epidemics of streptococcal, meningococcal and bacillary dysenteric infections have been brought under control readily by the prophylactic use of appropriate sulphonamides. They have proved useful in preventing secondary respiratory infections in certain virus diseases such as measles. For these purposes the drugs are used in doses approximately half of those used in treatment of the established disease of moderate severity.

The prolonged use of small doses (0.5 to 1.0 gramme daily) of these drugs for the prevention of the streptococcal infections so frequently preceding relapses of rheumatic fever

has been very successful in reducing the incidence of such relapses, though the oral use of penicillin is at least equally reliable.

During prolonged prophylactic therapy, a watch must be kept for toxic effects, which are fortunately rare. There is also a definite risk of development of resistant strains of organisms under these circumstances. But this method of preventing relapses in rheumatic fever has proved so successful with both sulphonamides and penicillin that its use should be most seriously considered for every child with rheumatic fever.

KEITH D. FAIRLEY,
Melbourne.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held on February 28, 1952, in the Verco Theatre, Institute of Medical and Veterinary Science, Adelaide, the President, DR. R. L. THOROLD GRANT, in the chair.

Psychiatric Disorders in General Practice.

DR. H. M. BIRCH read a paper entitled "Psychiatric Disorders in General Practice" (see page 813).

DR. W. M. WIGG opened the discussion. He said that rarely had they heard a paper of such interest and importance as the one which Dr. Birch had just presented. Whatever branch of medicine they followed there was something in it for all, and he would like to congratulate Dr. Birch not only on the choice of material, but also on his clear and concise mode of presentation. Many of the audience had acquired for the first time, perhaps, something like a clear conception of the early schizophrenic. What matter if some of them, himself included, now saw themselves in their true psychological colours?

Dr. Wigg went on to say that the part of Dr. Birch's paper dealing with certification had stirred their consciences, and about time, too. He was especially interested to learn of the new aid that might now be expected from the police in certain circumstances. Such help would have saved him much anxiety on at least two occasions many years previously in lonely farm-houses, when attacked or threatened by homicidal maniacs; "anxiety" was possibly an under-statement.

Before commenting on the rest of Dr. Birch's paper, Dr. Wigg said that he would like to touch on a few of the psychiatric problems one met with in general practice. As the older practitioners had already learned the answers to many of the problems, his remarks were directed mainly to those embarking on practice in whatever their chosen sphere. He hoped that they would criticize the comments, as they were offered solely to promote discussion. The problem of the early senile dement was always present. Personally Dr. Wigg was reluctant to send that patient to hospital until it was quite inevitable. In their lucid intervals their distress at change of environment was often pitiable, and could be a factor, he thought, in hurrying their downward progress. One could usually postpone the day by persuasion of relatives, and by the use of suitable drugs, of which he had found carbromal ("Carbital") the one of choice. It was to be remembered, too, that in his normal interludes the early senile dement had normal feelings, and should be treated accordingly.

A matter that must not be overlooked was the fear factor in obstetrics. While all might not be firm disciples of Grantly Dick Read, it had to be admitted that he had got something. At any rate it was their bounden duty to give an explanatory talk to every *primipara* before her friends and relatives got at her. Her labour would be the easier for it.

There was not time to consider the problem of the difficult child, beyond pointing out that the child's whole future might depend on correct handling of the situation. Suffice it to say that in the main doctors must treat the parent rather than the child, and leave the treatment of the child to other children. That, of course, was true only in the broad sense, and Dr. Wigg would like to hear some discussion on the problem. Just in passing, Dr. Wigg mentioned the miraculous cures that one could occasionally effect in the child

who had nightmares by giving him a glucose drink at bedtime. So often he was hypoglycemic from overfatigue.

To promote later discussion Dr. Wigg said that he was going to make the statement that duodenal ulcer had a wholly psychogenic origin (except for the small part possibly played by tobacco in a few cases) and that ulcerative colitis was almost in the same class. He was aware that he was "sticking his neck out" making that statement, but he believed it to be true. As yet he had failed to find a case of either which did not have a fairly easily discernible underlying emotional element, and if that was removed amelioration or cure of the condition followed.

Dr. Wigg's remaining comments concerned the subject of the first part of Dr. Birch's paper, the common neuroses. Dr. Wigg paid tribute to his friend and partner, the late Dr. John Gillen, who was, he said, a past master in handling such patients. From him he had acquired an abiding interest in them. However, any over-emphasis he might have come to place in his early years on the psychiatric factors in disease had received a setback later in out-patient hospital practice, where he became rather depressed at the number of patients whom they thought to have been investigated fully, that later, on reinvestigation, revealed an obvious physical cause for their symptoms. But what was even worse, when they applied treatment that should have relieved the symptoms, not infrequently the patient persisted in his complaints regarding them, or showed other evidence of psychological ill. They had been too late in their detection of the physical ill. That sort of experience, common no doubt in its essentials to all, brought him to emphasize three points. The first was the vital importance of complete physical examination as a prerequisite to the investigation or treatment of any psychiatric patient. Dr. Birch had already drawn their attention to that. But one must not be afraid to review the diagnosis if necessary—the obvious psychiatric disease might be only a part of the whole. The second point was the frequency with which the apparently most straightforward case had a psychiatric facet; to mention only one obvious example, the woman whose vomiting of pregnancy did not respond to simple treatment, and in whom one found the cause among a wide variety of emotional disturbances. The third point was that the time to commence investigation of the psychiatric aspect was the moment it was suspected, and the time to commence its treatment was the moment the patient had revealed to the doctor his inner mind. Dr. Wigg did not mean that they should commence a mental onslaught on the patient at the first interview; often a mere touching on the psychological aspect then would make it possible to gain the truth at the next consultation. His point was that once one had the inside story one must immediately offer the patient a way out of his difficulties if possible, or at least explain clearly the origin of his apparently real symptoms. When the case was mainly somatic, a simple explanation on those lines might be all that was necessary. Patients were extremely grateful for such explanations, and failure to give them was one cause of that tragic progress which some dissatisfied patients made from practitioner to practitioner, or from specialist to specialist, and which could so discredit the profession.

In all cases the time factor was vital, and Dr. Wigg said that he would regard the state of the patient in the moment when he was about to reveal, or had just revealed, his innermost secrets to the doctor as a psychiatric emergency no less urgent than its surgical counterpart, the "acute abdomen", and the patient was no less liable to disaster if mishandled. Some such situations were comparable perhaps to the airman's "point of no return". Should the doctor fail in his duty there, the patient might never again feel disposed to confide in him or perhaps even in anybody else. At best the doctor might cure him there and then, and his gratitude would sustain the doctor in the stresses that awaited him in the form of angry patients whom he had kept waiting for an hour. At worst, the doctor would realize that he was dealing with material that was unlikely to respond to simple psychiatric treatment, and he could at least take early steps to place the patient in expert hands. Given time, they all acquired, if they were interested, a measure of low cunning in such matters. But, speaking of his own generation at least, Dr. Wigg asked if they should not have commenced their years of practice a little better equipped. He heartily agreed with Dr. Birch's comments in that regard. The teaching of elementary psychiatry, and the study of the individual's personality, should begin in the general hospital in every ward, at every bedside, and at every out-patient consultation.

Finally, Dr. Wigg admitted to a fear that the rising tide of socialized medicine would make it almost impossible for

the doctor to do his duty to his patients. It was difficult enough at present to find the time. Yet was not a man's happiness of more importance to him and to his doctor than a self-curtative bodily ill? Still, no doubt the general practitioners' arteries would hustle them off before they were quite submerged in forms, and could no longer do anything for the victims of the conditions that they had discussed.

DR. A. R. SOUTHWOOD remarked on the better attention given to neuroses and psychoses in present-day clinical teaching compared with the hesitancy of general physicians twenty years or so previously to face the problems. He said that then it was common in the wards to hear the remark, "This is only neurosis", as the teacher and his group passed quickly to the next patient. In the Adelaide Hospital the late Dr. Frank Hone had constantly directed students' attention to the importance of the psychic factor in disease, and to the need for careful case-taking with that fact in mind. The book by T. A. Ross, "The Common Neuroses", was a valuable help to physicians when it appeared in 1920 or so. Now one talked glibly of psychosomatic medicine, as if it were altogether new. Physicians in the past were often urged to "know syphilis", so common it was and so protean were its features. Today a better piece of advice would be to "know psychiatry".

DR. R. D. HORNABROOK said that he would like to support Dr. Wigg in his stressing of the importance of being prepared to make the most of that rare occasion when a patient came prepared to tell the doctor what might be the clue to the whole illness. That was well illustrated by the case of a young woman who came complaining of headaches following concussion while on holiday. She had been in a country hospital for a day or two and then returned to her caravan with swimming and all her usual activities. Her headaches lasted for months, during which time she developed epileptiform convulsions. She was examined by a neurosurgeon on two occasions, when no definite cause could be found. After Dr. Hornabrook performed a lumbar puncture on her in hospital she complained of headache, nausea and vomiting, and giddiness for over a week. The spinal fluid was normal in every way. She eventually recovered, but had not been long at work when she had frequent bowel actions, passing bright blood *per rectum*. Sigmoidoscopy showed that she had acute ulcerative colitis, which settled down quickly on sedation in hospital. On return to her work she was again troubled with headaches, "blackouts" and "fits". In spite of numerous conferences with her mother and father and the patient, it was not until what would have quite likely been her last visit to Dr. Hornabrook that she told him the true position at home. Her father had financed her in her business, but, she said, in such a way that not only could she not make any return for herself, but by her working harder her overhead increased and she appeared to be working herself into debt. On her cutting her losses—selling the business and working for one of the big firms in town—her trouble cleared up. Dr. Hornabrook remarked that while she was telling him all this, his other appointments were getting an hour or so behind, but he thought that his audience would agree that the result was worth it.

DR. DOUGLAS SALTER stressed the importance of trying to ascertain whether a psychiatric patient was a potential suicide. If he was considered so, he should not be left alone while arrangements were being made for him to be admitted to hospital, as a person contemplating committing suicide was especially likely to do so at that time. Dr. Salter said that on two occasions he had had patients shoot themselves under those conditions. He also mentioned the fact that elderly people, especially if they lived by themselves and were on an inadequate diet, might have symptoms mistaken for senile dementia, whereas they were suffering from vitamin deficiency.

DR. D. A. HICKS said that there was a definite development of organic alimentary tract lesions in those cases previously labelled functional or abdominal neurosis. Many patients had been examined repeatedly by barium meal *et cetera* and had at last shown definite evidence of, for example, duodenal ulcer not found in the earlier stages. That seemed significant, even allowing for the pitfalls in radiological diagnosis. Dr. Hicks went on to say that explanation and reassurance for the patient were helpful in many cases and certainly an improvement on the old attitude of "nothing wrong, only your nerves". But it was most important to avoid misunderstanding and subsequent antagonism by pre-fixing the remarks by emphasizing that one was not accusing the patient of malingering, or criticizing him, or implying that he was inadequate (although at times that might be the real answer), and by explaining that in many cases the symptoms were normal to everyone, but that attention

was more readily focused on them and they assumed greater significance under conditions of tension.

DR. E. F. GARTRELL expressed his appreciation of Dr. Birch's address and stressed the importance of remembering, as had been pointed out by the speaker, that there frequently was an overlay of anxiety in the presence of organic lesions. He said that Dr. Wigg's emphasis on the necessity for taking adequate time in investigating the psychoneuroses was not misplaced and also added to his own conviction that in all medical cases a good history was of paramount importance. This was exemplified in two cases recently seen in consultation. The first was that of a girl of twenty-two years suffering from a paroxysm of ventricular tachycardia with a rate of 250 per minute. No abnormality of the heart could subsequently be demonstrated, but had quinidine not been effective death would have resulted from the condition which was partly, if not wholly, of emotional origin. Investigation of the patient's history disclosed two nervous breakdowns. In addition it was curious that the father was present at the examination and that the mother came in only at the end to tidy up. Further interrogation would probably have led to an underlying cause in the shape of domestic difficulty.

The second case was one in which an attack of tachycardia with a rate of 180 per minute was accompanied by epigastric discomfort *et cetera* in a woman of sixty-five years. This proved to be due to auricular fibrillation, but what was underlying it? There was no evidence of coronary occlusion, rheumatic heart lesions, hypertension or thyrotoxicosis. Slight T wave changes were quite equivocal. Further questioning brought to light a fierce family argument immediately prior to the onset of symptoms. The emotional factor had precipitated important functional change.

Dut of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

AN APPLICATION FOR FORAGE ALLOWANCE.¹

Colonial Secretary's Office,
Sydney, February 27, 1835.

James Bowman, Esq.
Sir,

Having laid before the Governor your letter of 14th Instant repeating the application for Forage for Mr Surgeon Mitchell, I am directed to inform you, that His Excellency sees no reason for allowing this contingency to more than one of the medical officers attached to the Sydney Hospital, who should take all the exterior duties.

I am further directed to inform you, with reference to the last paragraph of your letter, that His Excellency considers the apprehension, therein expressed, of the consequence of withholding the Forage allowance, as altogether gratuitous.

I have, &c.,
ALEX. MCLEAY.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Hallstrom Institute of Cardiology, Royal Prince Alfred Hospital.

The second annual post-graduate course conducted by the Hallstrom Institute of Cardiology, in conjunction with the Post-Graduate Committee in Medicine, will be held in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, from July 14 to 25, 1952. The course will consist of a series of ten afternoon lectures followed by demonstrations of patients and other relevant material. Emphasis will be placed this year on the modern theory, practice and inter-

¹ From the original in the Mitchell Library, Sydney.

pretation of electrocardiograms, and this section of the course will be open to electrocardiograph technicians employed by hospitals and the armed services.

The lectures and demonstrations will be conducted by the staff of the Institute, and ample opportunity for discussion will be provided. Visitors will also be able to participate in two of the regular post-graduate medical afternoons arranged by the Department of Medicine, Royal Prince Alfred Hospital.

Those who wish to attend this course, and who are not members of the staff of the Royal Prince Alfred Hospital, must register with the Course Secretary, Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. The fee will be £6 6s.

Lectures by Professor G. F. Gibberd.

In conjunction with the Royal College of Obstetricians and Gynaecologists, Professor G. F. Gibberd, M.B., M.S., F.R.C.S., F.R.C.O.G., Senior Obstetric Surgeon at Guy's Hospital and first Sims-Black Professor, will give two lectures during his visit to Sydney, on July 22 and 24. Those wishing to attend are asked to notify the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 6238, BW 7488.

Week-End Course at Katoomba.

The Post-Graduate Committee in Medicine in the University of Sydney announces that, in conjunction with the Blue Mountains Medical Association, a week-end course will be held at Katoomba (in the ballroom of the Carrington Hotel) on Saturday and Sunday, June 28 and 29, 1952. The programme is as follows:

Saturday, June 28: 2 p.m., registration; 2.30 p.m., "Problems of Antibiotic Therapy", Dr. Edgar Thomson; 4 p.m., "Surgical Exposures", Dr. P. J. Kenny.

Sunday, June 29: 10 a.m., "Common Disorders of Gynaecology, I", Dr. Malcolm Stening; 11.30 a.m., "Recent Advances in Clinical Pathology", Dr. Edgar Thomson; 2.30 p.m., "Fluid and Electrolyte Balance", Dr. P. J. Kenny; 4 p.m., "Common Disorders of Gynaecology and Obstetrics, II", Dr. Malcolm Stening.

Fee for attendance is £2 2s. Those wishing to attend are requested to communicate as soon as possible with Dr. Nicholas Larkins, Honorary Secretary, Blue Mountains Medical Association, Katoomba Street, Katoomba.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 36, of May 15, 1952.

AUSTRALIAN MILITARY FORCES.

Royal Australian Army Medical Corps.

NX700304 Captain (Temporary Major) R. Cuttle relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District), 20th February, 1952.

The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District) in the honorary rank of Captain: VX700245 P. E. Silberstein, 2nd February, 1952, and VX700230 J. S. Crosbie, 12th February, 1952.

To be Captain, 14th January, 1952.—3/40095 Ian Allan Le Gay Ferguson.

To be Captains (provisionally).—QX700138 Carol Lindsay Cleland and QX700137 Alexander Montague Horner, 14th January, 1952, QX700143 Murray Gowan Williams, QX700144 Graeme Bruce Roberts and VX700263 Gerald Redmond Garrett Prendiville, 11th February, 1952, QX700152 James Robert Syme and QX700151 Geoffrey Spencer Briggs, 10th March, 1952.

To be Captain, 14th January, 1952, with a Short Service Commission for a Period of One Year.—3/40095 Ian Allan Le Gay Ferguson (Captain).

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps.—1/61769 Honorary Captain L. J. Lowth is appointed from the Reserve of Officers, and to be Captain (provisionally), 26th February, 1952. 1/39084 Captain (provisionally) M. H. Lake relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District) in the rank of Honorary Captain, 7th February, 1952.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps.—2/127017 Captain (provisionally) W. C. Woods relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District) in the honorary rank of Captain, 18th February, 1952.

Southern Command: Third Military District.

Royal Australian Army Medical Corps.—3/50011 Colonel C. W. Ross relinquishes the appointment of Colonel, Advisory Committee (Medical Equipment), Director of Medical Services, "A" Branch, Army Headquarters, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District), 4th March, 1952. 3/50147 Major E. R. Crisp is appointed from the Reserve of Officers, is appointed Consultant Radiologist, Director of Medical Services, "A" Branch, Army Headquarters, and to be Temporary Colonel, 4th March, 1952. 6/15342 Captain (provisionally) J. F. Banfield relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District) in the honorary rank of Captain, 12th January, 1952. 8/101806 Captain (provisionally) G. M. Stubbs relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District) in the honorary rank of Captain, 17th January, 1952. To be Captain (provisionally), 23rd February, 1952: 8/101818 William Nicol Sloan.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps.—The following officers cease to be seconded for post-graduate studies in the United Kingdom: Captains 4/81905 G. W. E. Aitken, 17th December, 1951, and 4/81907 R. A. Burston, 7th January, 1952.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps.—The provisional appointment of 5/26398 Captain H. S. Cohen is terminated, 31st October, 1951. 5/26399 Captain (provisionally) H. S. Cohen is seconded whilst undergoing post-graduate studies in the United Kingdom, 1st November, 1951. The regimental seniority of 5/32255 Captain M. Mayrhofer is next after 5/26394 Captain (Honorary Major) H. J. Rowe. The following officers are appointed from the Reserve of Officers: 5/88060 Captain T. Godlee, M.B.E., 3rd January, 1952, and 5/21508 Captain (Honorary Major) M. G. F. Donnan, 21st January, 1952. 5/26466 Captain (provisionally) G. I. Howard relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District) in the honorary rank of Captain, 28th February, 1952. To be Captain (provisionally): 5/26395 Henry Sweetman Cohen, 1st November, 1951, with regimental seniority next after 5/26397 Captain (provisionally) W. I. Gordon, and 5/26507 Neil Same, 5/26508 Roy Allen, and 5/26509 Hal John Hester Colebatch, 3rd March, 1952.

Tasmania Command: Sixth Military District.

Royal Australian Army Medical Corps: To be Captain (provisionally), 22nd February, 1952.—6/20003 Hubert Desmond O'Brien.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District.—Captain N. B. Wilmer is granted the honorary rank of Major, 26th March, 1952. To be Honorary Captains, 18th March, 1952: Murray Scott Marshall and Frederick Schubert.

2nd Military District: To be Honorary Captains.—Robert Keith Reid, 6th March, 1952, and Geoffrey King Bernays, 28th March, 1952.

3rd Military District: To be Honorary Captain, 13th March, 1952.—Mary Ethel Thornton.
6th Military District.—Honorary Captain E. Fabian is retired, 29th February, 1952.

Public Health.

STARCH POWDER FOR SURGEONS' GLOVES.

THE following statement has been supplied by Dr. E. Sydney Morris, Director-General of Public Health for New South Wales.

Following receipt of a report from the Queensland Department of Health that tetanus bacilli had been found in prepared starch powder used in that State in the preparation of surgeons' gloves for surgical operations, investigations were carried out by the New South Wales Department of Public Health.

Twelve samples of starch powder were submitted for examination. These samples were not claimed to be sterile, and all but one gave a heavy growth on culture. The organisms grown proved to be aerobic and anaerobic sporing bacilli, and *Clostridium tetani* was isolated from one sample. Varying depths of starch powder were placed in flat-bottomed Petri dishes or beakers, and were treated in an autoclave, at 15 pounds' pressure for thirty minutes, with the object of determining whether such pressure would produce sterility.

It was found that sterility was obtained up to half an inch, but greater depths were not sterilized.

If an autoclave is to be used for sterilization, it is suggested that the depth of powder should not be greater than a quarter of an inch, and that the pressure should not be below 15 pounds per square inch and the time not less than thirty minutes.

Alternatively, the starch powder may be sterilized by dry heat in two hours, at a temperature of not less than 320° F.

Australian Medical Board Proceedings.

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners.

Clarke, Marcus C., M.B., B.S., 1937 (Univ. Sydney), D.T.M., 1946, M.R.C.S. (Edinburgh), 1951; Kinny, Noel Wesley, M.B., B.S., 1951 (Univ. Sydney); Petrovitch, Constantin C., M.B., B.S., 1938 (Univ. Hong Kong).

NEW SOUTH WALES.

THE following have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1950, of New South Wales, as duly qualified medical practitioners.

Adams, Warwick Leslie, M.B., B.S., 1952 (Univ. Sydney); Adecock, Vincent Joseph, M.B., B.S., 1952 (Univ. Sydney); Anderson, Peter Francis, M.B., B.S., 1952 (Univ. Sydney); Andreazza, Joyce, M.B., B.S., 1952 (Univ. Sydney); Armstrong, Philip John, M.B., B.S., 1952 (Univ. Sydney); Arnold, Dennis Deane, M.B., B.S., 1952 (Univ. Sydney); Auerbach, Egan, M.B., B.S., 1952 (Univ. Sydney).

Barnett, John Sadler, M.B., B.S., 1952 (Univ. Sydney); Barton, David Edmund, M.B., B.S., 1952 (Univ. Sydney); Bassar, Philip Nathan, M.B., B.S., 1952 (Univ. Sydney); Bassett, Ray, M.B., B.S., 1952 (Univ. Sydney); Beith, John McNeil, M.B., B.S., 1952 (Univ. Sydney); Bennett, James Warren Bruce, M.B., B.S., 1952 (Univ. Sydney); Bennett, Pamela Marjorie, M.B., B.S., 1952 (Univ. Sydney); Bennaya, Geoffrey King, M.B., B.S., 1952 (Univ. Sydney); Bingham, Elaine Edith, M.B., B.S., 1952 (Univ. Sydney); Boa, Peter Alfred, M.B., B.S., 1952 (Univ. Sydney); Booth, Molle, M.B., B.S., 1952 (Univ. Sydney); Borger, James Paul, M.B., B.S., 1952 (Univ. Sydney); Borkman, Helmo, M.B., B.S., 1952 (Univ. Sydney); Bourke, Geoffrey Merwin, M.B., B.S., 1952 (Univ. Sydney); Boyd, Brian Anthony Majella, M.B., B.S., 1952 (Univ. Sydney); Bradbury, Roger, M.B., B.S., 1952 (Univ. Sydney); Brown, Bruce Alexander, M.B., B.S., 1952 (Univ. Sydney); Brown, Richard Francis, M.B., B.S., 1952

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 17, 1952.¹

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Arnhem Land.
Acute Rheumatism
Amebiasis
Ancylostomiasis
Anthrax
Bilharziasis
Brucellosis
Cholera
Chorea (St. Vitus)
Dengue
Diarrhoea (Infantile)	15(5)	2	24(24)	..	1(1)	..	1	..	26
Diphtheria	..	1(1)	15(8)	32
Dysentery (Bacillary)	..	2	6(6)	1(1)	2(2)	10
Encephalitis	2
Malaria
Homologous Serum Jaundice
Hydatid	..	1	1
Infective Hepatitis	5
Lead Poisoning
Leprosy
Leptospirosis	2
Malaria
Meningococcal Infection	5(8)	3(8)	2(1)	1	1	4	15
Ophthalmia
Otitis
Paratyphoid	1(1)	1
Plague
Pollomyelitis	..	6(3)	6(4)	3(2)	4(4)	1	26
Periperal Fever
Rubella	2(2)	1(1)	..	1	4
Salmonella Infection	1(1)	..	3	4
Scarlet Fever	20(8)	27(16)	1(1)	8(7)	5(5)	3	64
Smallpox
Tetanus
Trachoma
Trichinosis
Tuberculosis	29(23)	26(13)	21(12)	7(4)	19(18)	2(1)	..	2	99
Typhoid Fever
Typhus (Flea, Mite, and Tick-borne)
Typhus (Louse-borne)
Yellow Fever

¹ Figures in parentheses are those for the metropolitan area.

(Univ. Sydney); Bruce, Douglas William, M.B., B.S., 1952 (Univ. Sydney); Bryce, Beverley Wynifred, M.B., B.S., 1952 (Univ. Sydney); Buckingham, William, M.B., B.S., 1952 (Univ. Sydney); Bullen, Monica Mary, M.B., B.S., 1952 (Univ. Sydney); Burnham, Rhonda, M.B., B.S., 1952 (Univ. Sydney); Burns, David, M.B., B.S., 1952 (Univ. Sydney); Burnside, Colin Campbell, M.B., B.S., 1952 (Univ. Sydney); Bushell, Douglas Ian, M.B., B.S., 1952 (Univ. Sydney).

Calov, Walter Tom, M.B., B.S., 1952 (Univ. Sydney); Campbell, Donald McLeod, M.B., B.S., 1952 (Univ. Sydney); Carey, Enid Menary, M.B., B.S., 1952 (Univ. Sydney); Chalmers, Clair Drummond, M.B., B.S., 1952 (Univ. Sydney); Chapman, Maxwell Graham, M.B., B.S., 1952 (Univ. Sydney); Chapman, Walter Raymond, M.B., B.S., 1952 (Univ. Sydney); Chappel, Roy Alfred, M.B., B.S., 1952 (Univ. Sydney); Church, David Thomas, M.B., B.S., 1952 (Univ. Sydney); Clarence, Leslie Norman, M.B., B.S., 1952 (Univ. Sydney); Clifton, Bruce Stewart, M.B., B.S., 1952 (Univ. Sydney); Coleman, John, M.B., B.S., 1952 (Univ. Sydney); Collier, Beryl, M.B., B.S., 1952 (Univ. Sydney); Collins, Kenneth Leslie, M.B., B.S., 1952 (Univ. Sydney); Collison, Jeanne Margaret, M.B., B.S., 1952 (Univ. Sydney); Connolley, Thomas Joseph Bede, M.B., B.S., 1952 (Univ. Sydney); Connolly, John Anthony, M.B., B.S., 1952 (Univ. Sydney); Cook, Walter Arthur Aufre, M.B., B.S., 1952 (Univ. Sydney); Coorey, Kevin Dominic Francis, M.B., B.S., 1952 (Univ. Sydney); Cope, Barbara Stewart, M.B., B.S., 1952 (Univ. Sydney); Cordingley, John Louis, M.B., B.S., 1952 (Univ. Sydney); Cornfield, William Harry, M.B., B.S., 1952 (Univ. Sydney); Coster, Alwyne Reta, M.B., B.S., 1952 (Univ. Sydney); Cotton, Keith Lucas, M.B., B.S., 1952 (Univ. Sydney); Cousins, Adrian Gordon, M.B., B.S., 1952 (Univ. Sydney); Coyne, Francis John, M.B., B.S., 1952 (Univ. Sydney); Crawshaw, Brian Keith, M.B., B.S., 1952 (Univ. Sydney); Croll, Francis James Thomas, M.B., B.S., 1952 (Univ. Sydney); Crooks, Royle Reuben, M.B., B.S., 1952 (Univ. Sydney); Cumming, Robert Wellington, M.B., B.S., 1952 (Univ. Sydney); Cutler, Geoffrey Abbott, M.B., B.S., 1952 (Univ. Sydney).

D'Arbon, Paul John Peter, M.B., B.S., 1952 (Univ. Sydney); Dash, Edward Geoffrey, M.B., B.S., 1952 (Univ. Sydney); Davis, Joseph Ivan, M.B., B.S., 1952 (Univ. Sydney); Dawson, Bruce Hamilton, M.B., B.S., 1952 (Univ. Sydney); De Launey, Wallace Edward, M.B., B.S., 1952 (Univ. Sydney); De Meyrick, George William, M.B., B.S., 1952 (Univ. Sydney); Dickens, Brian Forster, M.B., B.S., 1952 (Univ. Sydney); Diethelm, Geoffrey, M.B., B.S., 1952 (Univ. Sydney); Dillane, John Bevan, M.B., B.S., 1952 (Univ. Sydney); Dinning, Graham Gordon, M.B., B.S., 1952 (Univ. Sydney); Dodd, Grahame Powis, M.B., B.S., 1952 (Univ. Sydney); Donnelly, George Lawrence, M.B., B.S., 1952 (Univ. Sydney); Donovan, John Kenmore, M.B., B.S., 1952 (Univ. Sydney); Dorner, John Cecil, M.B., 1952 (Univ. Sydney); Doust, Kenneth McGowen, M.B., B.S., 1952 (Univ. Sydney); Dow, Edward Fitzherbert, M.B., B.S., 1952 (Univ. Sydney); Doyle, Francis, M.B., B.S., 1952 (Univ. Sydney); Drummond, Norman Cranmer, M.B., B.S., 1952 (Univ. Sydney); Duke, John Swinnerton, M.B., B.S., 1952 (Univ. Sydney); Dunn, Robert Harper, M.B., B.S., 1952 (Univ. Sydney); Dyce, John James Douglas, M.B., B.S., 1952 (Univ. Sydney); Dykes, John Dudley, M.B., B.S., 1952 (Univ. Sydney).

Honours.

BIRTHDAY HONOURS.

THE following are included among the Birthday Honours conferred by Her Majesty the Queen:

Dr. Hugh Raymond Guy Poate, of Sydney, has been created a Knight Bachelor.

Dr. John Joseph Holland has been created a Commander of the Most Excellent Order of the British Empire.

Medical Appointments.

THE following have been appointed, under the provisions of the Anti-Cancer Council Act, 1936, members of the Anti-Cancer Council of Victoria: Dr. Henry Searby, Dr. J. E. C. Clarke, Dr. K. Hiller, Dr. Leo Doyle, Dr. B. Quick, Dr. L. S. Kidd, Dr. T. E. Lowe, Professor Sir Macfarlane Burnet, Dr. C. A. M. Renou, Dr. D. J. Thomas, Dr. D. B. Pearce, Sir John Newman-Morris, Dr. Robert Fowler, Dr. R. Kaye Scott.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

O'Neill, John Patrick, M.B., B.S., 1948 (Univ. Sydney), 16 Lang Road, Centennial Park.
Bradley, Edgar David, M.B., B.S., 1951 (Univ. Sydney), Maitland Hospital, Maitland.
Cahalan, Bernard Joseph Seymour, M.B., B.S., 1946 (Univ. Sydney), Mental Hospital, Orange.

Diary for the Month.

JUNE 16.—Victorian Branch, B.M.A.: Finance Subcommittee.
JUNE 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 18.—Western Australian Branch, B.M.A.: General Meeting.
JUNE 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 19.—Victorian Branch, B.M.A.: Executive Committee.
JUNE 19.—South Australian Branch, B.M.A.: Scientific Meeting.
JUNE 24.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 125 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victoria Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desirous to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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